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THIS NUMBER CONTAINS PAPERS READ AT THE FIRST BIENNIAL MEETING OF THE FAR EASTERN ASSOCIATION OF TROPICAL MEDICINE, HELD AT MANILA, MARCH 5 TO 14, 1910.

THE PHILIPPINE

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B. MEDICAL SCIENCES

VOL. V JULY, 1910 No. 2

STUDIES ON INFANT MORTALITY.[1]

By ALLAN J. MCLAUGHLIN[2] and VERNON L. ANDREWS.[3]

The death rate among Filipinos in Manila, as shown below, is excessive compared with that of other nationalities, after making due allowance for the higher birth rate, greater proportion of children, and other factors.

TABLE I.—*Death rate in Manila by nationalities, per thousand, during the year 1909.*

Spaniards	12.05
Americans	13.27
Other Occidentals	14.32
Chinese	16.64
Filipinos	47.65

This enormous death rate is due to the high mortality of children. Of 9,307 deaths among the Filipino population, 6,041, or 64.9 per cent, were of children under 5 years of age, and 4,542, or 48.8 per cent, were of infants under 1 year of age.

TABLE II.—*Rate of infant mortality to total number of deaths.*

Total number of deaths, all ages	9,307
Total number of deaths under 5 years	6,041
Total number of deaths under 1 year	4,542
Rate of deaths under 1 year to total number, per cent	48.8

[1] Read at the First Biennial Meeting of the Far Eastern Association of Tropical Medicine, March 9, 1910.

[2] Passed Assistant Surgeon, United States Public Health and Marine-Hospital Service; Assistant Director of Health for the Philippine Islands; and assistant professor of Hygiene, Philippine Medical School.

[3] Assistant, Biological Laboratory, Bureau of Science, and Assistant Professor of bacteriology and pathology, Philippine Medical School.

Compare this infant mortality with that of other countries and the result is striking. According to the United States census of the year 1900, the deaths of children under 1 year constitute 18.28 per cent of the total mortality; in France,[4] from 1896 to 1900, the infant mortality constituted 20 per cent of the total, but in Manila the deaths of infants under 1 year aggregate 48.8 per cent of the total number.

TABLE III.—*Rate of deaths of children to total number of deaths, in percentages.*

United States	18.28
France	20.00
Manila	48.8

In order to combat intelligently this appalling infant mortality, it is essential to determine accurately what diseases really constitute the greatest factors and these investigations were undertaken with that end in view.

According to the statistics of the Bureau of Health, the main factors in child mortality for the fiscal year 1908–9, a fairly representative year, were the following:

TABLE IV.—*Factors in the mortality of children in Manila.*

Cause of death.	Number of deaths, children under 1 year.	Number of deaths, children under 5 years.
Convulsions	1,615	1,615
Congenital debility	596	596
Beriberi	595	629
Acute bronchitis	569	689
Acute meningitis	287	510
Enteritis	286	745
All other causes	594	1,257
Total	4,542	6,041

It was necessary to verify these statistics. The figures for the total number of deaths were undoubtedly correct, but the causes given in the death certificates were demonstrated frequently to be incorrect and indefinite.

In a previous paper, one[5] of us presented some observations upon cholera in children during the period from August 1 to September 25, 1909, and among the conclusions deduced from those observations were the following:

1. Cholera in children is often unrecognized and unreported as such, the diagnosis being reported as acute or chronic enteritis, gastroenteritis, enterocolitis,

[4] Budin. *Ann. de Med. et chir. infant* (1903), 7, 181.

[5] McLaughlin. Some Observations Upon Cholera in Children. *This Journal, Sec. B* (1909), 4, 363.

dysentery, acute or simple meningitis and probably also as infantile beriberi, convulsions of children, and some other forms of disease.

2. Cholera in children is often atypical, and in these cases diagnosis is extremely difficult, if not impossible, without bacteriological examination of the intestinal contents.

3. Cerebral manifestations in children suffering from cholera are very common and their severity is in inverse proportion to the age of the child.

4. Acute meningitis is a very rare disease in Manila, in spite of statistics.

5. The percentage of children attacked by cholera is higher than is shown by statistics.

In our work we are able to confirm the conclusions expressed above in regard to meningitis and cholera, and to present additional data bearing upon infantile beriberi, convulsions of children, enteritis, bronchitis and broncho-pneumonia. The clinical history as gleaned from the family or attending physician is given in the tables. These histories are meager and are not always reliable, owing to the fact that the case is often seen late in the disease and to the difficulty of getting correct data from the family. However, it is unlikely that all the histories are unreliable, particularly as the unanimity is so marked.

In all cases of meningitis, beriberi, convulsions and bronchitis the necropsy findings are given. In the cases of enteritis and dysentery, necropsies were not performed in every instance, and confirmation of the diagnosis of Asiatic cholera was secured by bacteriologic examination of the intestinal contents at the Bureau of Science.

ACUTE MENINGITIS.

The following table shows the deaths from acute meningitis in Manila by months for the past nine years:

TABLE V.—*Acute meningitis in Manila from 1901 to 1909, inclusive.*

Month.	1901	1902	1903	1904	1905	1906	1907	1908	1909
January	26	21	17	25	28	22	23	78	33
February	24	21	14	26	16	23	23	92	32
March	56	28	17	24	26	30	22	44	34
April	21	25	14	25	34	32	28	31	27
May	29	50	30	32	24	35	37	35	24
June	22	54	13	47	27	40	23	45	48
July	15	34	17	51	31	95	27	93	31
August	27	39	36	40	34	70	34	64	40
September	33	25	43	49	58	40	58	62	27
October	28	34	41	50	32	25	58	54	4
November	26	29	33	30	24	26	43	46	2
December	25	32	26	36	21	30	50	45	3
Total	331	392	301	435	358	468	421	689	305

Average number of cases per year, 410; average number of cases per month, 34.

These statistics cover nine full years, and from them it will be noticed that the average number of deaths from meningitis in Manila was about 410 per year, or 34 per month. The investigation by necropsy of children dead from alleged meningitis began the last week of September, 1909. The number of deaths from meningtis recorded for October dropped to 4, for November to 2, and for December to 3.

Table VI, opposite page 160, shows the cases of alleged acute meningitis which came to necropsy, together with the clinical data and pathologic findings.

The following is a summary:

Summary of Table VI.

Acute meningitis	2
Pneumonia	2
Empyema	1
Beriberi	10
Cholera	18
Undetermined (not meningitis)	3
Enterocolitis	1
Total	37

ACUTE ENTERITIS AND OTHER DIARRHŒAS.

Tabuc VII, opposite page 160, gives an analysis of a number of cases of alleged enteric disease.

We wish to emphasize the fact that diagnosis in these cases was difficult, or impossible from the clinical symptoms and that the examination of the intestinal contents should be compulsory in all children acutely sick in Manila. In these instances the classical symptoms of cholera were either absent or overlooked, because of their transient character. Sometimes the choleraic signs present were masked by intercurrent diseases and the predominance of cerebral manifestations.

Summary of Table VII.

Cholera	15
Beriberi	2
Pneumonia	2
Enterocolitis	3
Total	22

INFANTILE CONVULSIONS.

The diagnosis "infantile convulsions" should not be accepted as a cause of death. It is only a symptom of some specific disease and has no more right to a place in statistics than fever or chills. Table VIII, opposite page 160, shows the results obtained from investigations by

necropsy of cases of alleged infantile convulsions, with clinical data and pathologic findings.

Summary of Table VIII.

Beriberi	31
Cholera	4
Pneumonia	1
Enterocolitis	1
Empyema	1
Cerebral hæmorrhage	1
Undetermined	1
Total	40

The importance of securing correct diagnoses in the cases reported as dying of infantile convulsions is at once evident in view of the fact that 35 per cent of the total mortality under 1 year is given on the death certificates as being due to this cause. In accepting these reports we admit that the real cause of 35 per cent of the mortality in Manila of infants under 1 year of age is unknown to us.

BRONCHITIS.

Table IX, opposite page 160, shows the results of necropsies on 27 cases in which the death certificate gave acute or chronic bronchitis, or bronchopneumonia as the cause of death. Some of the bodies were in bad condition when they came to autopsy, owing to warm weather supervening and the difficulty of transferring them to the morgue with sufficient promptness.

Summary of Table IX.

Beriberi	14
Pneumonia	6
Meningitis	2
Nephritis	2
Chronic colitis	1
Acute tonsilitis, pharyngitis and bronchitis	1
Undetermined	1
Total	27

INFANTILE BERIBERI.

Table X, opposite page 160, gives the results in cases of alleged infantile beriberi. The first cases of so-called infantile beriberi which we investigated in November proved to be of Asiatic cholera, and we were at that time somewhat doubtful of the existence of the former disease in infants and rather expected that our experience with alleged acute meningitis would be repeated. Further study showed that a large number

of infants die of a disease which presents a definite pathologic picture for which we know no better name than "moist beriberi."

Summary of Table X.

Beriberi	40
Cholera	3
Broncho-pneumonia	3
Enterocolitis	1
Undetermined	3
Total	50

In the pathological entity which we have called "beriberi" we come face to face with one of the real factors in Filipino infant mortality. This disease is responsible for many more deaths than would appear to be the case from statistics. We have found not only that many instances of alleged congenital debility and convulsions are due to beriberi, but also acute bronchitis and pneumonia. It is our opinion that the two diseases last mentioned are not common, and an investigation of deaths from acute bronchitis and broncho-pneumonia shows many of them to be due to beriberi.

The clinical symptoms of dyspnœa and cardiac embarrassment are often responsible for a diagnosis of bronchitis, or broncho-pneumonia, when the necropsy reveals the picture of beriberi. While we recognize the fact that bronchitis is a disease of childhood and old age, we hardly think that the figures as given in Table IV for bronchitis state the truth. It seems to us that the number of children under 1 year is too large and that the difference between 1 year and 5 is too small. True, it is in early infancy that the disease is most marked and it is more prevalent in squalid homes and among poorly nourished infants than elsewhere, yet we doubt if this would account for the figures as given.

A summary of the 176 cases studied is given in Table XI.

TABLE XI.—*Showing the number of cases investigated, together with clinical diagnoses and necropsy findings.*

Clinical diagnoses.		Necropsy findings.	
Meningitis	37	Cholera	40
Enteritis	22	Beriberi	97
Convulsions	40	Pneumonia	14
Beriberi	50	Enterocolitis	7
Bronchitis	27	Meningitis	4
		Nephritis	2
Total	176	Empyema	2
		Acute tonsilitis, pharyngitis and bronchitis	1
		Cerebral hæmorrhage	1
		Undetermined	8
		Total	176

The great discrepancy between the clinical diagnoses and the autopsy findings can partially be explained by the fact that in many instances the native doctor is not called to see the patient until the latter is moribund, or, in some cases, until after death, and the family want a death certificate signed.

In 1898 and 1900, Professor Hirota,[6] of Tokyo, described a disease found in some infants brought to his clinic, which he called infantile beriberi. A few years later, 1905 (?), the native doctors of Manila and the Philippines accepted Hirota's findings and began signing death certificates giving the cause of death as infantile beriberi. So far as the writers know, no effort was made by the native physicians to establish by necropsy any relation between the findings of Hirota in Tokyo and the disease called infantile beriberi by the Filipinos. For some reason, the subject has never been taken up by the American physicians in the Islands, owing, possibly, to the fact that they do not come in contact with the poorer Filipinos, and hence are never called upon to treat them. In the various hospital dispensaries the children are looked after by the native doctors. A number of Filipino physicians have recognized the condition here and one[7] has described a typical case with necropsy.

In the pathologic entity which we have termed "infantile moist beriberi," and for which we know no better name, we find the following conditions:

The body is that of an apparently well-nourished infant, plump; skin is usually pale and anæmic. The face is full, with almost a swollen appearance. Flesh of thighs and legs is soft and flabby and, as a rule, pits on deep pressure. Occasionally the skin has a tough, leathery feel, a leaden color and a slight goose-flesh appearance.

Subcutaneous fat is present, apparently in good amount, grayish-white in color and very moist; muscles are anæmic. Owing to the œdema, the real amount of fat present is deceptive and hence the bodies may not be as well nourished as they appear.

Most often there is an increase of peritoneal fluid, which has a distinct yellowish color.

Heart.—The pericardial sac is filled with a clear fluid, having a greenish tint. Probably the most striking and constant change is found in the right heart. Its musculature is coarse and firm and forms much the larger part of the organ, even in the contour of the apex. Its trabeculæ and papillary muscles are prominent, while its cavity is enlarged. The wall of the right ventricle may measure from 5 millimeters to 7 millimeters in thickness, whereas the left measures only 3 millimeters to 5 millimeters. (See Plate 1.) The musculature of the left heart is soft and flabby and darker than that of the right. The blood vessels of the heart are congested and prominent and frequently a few hæmorrhages show along the auriculo-ventricular junction. In many cases the foramen ovale is still patulous, but is competent.

Lungs.—These organs are a light pinkish-gray anteriorly and a light purplish-gray posteriorly. They fill the pleural cavities and crepitate throughout. The anterior part of the lung is lighter and more fluffy than the posterior. Few or many petechial hæmorrhages may show beneath the visceral pleura, especially

[6] *Centralbl. f. inn. Med.* (1898), 19, 385; *ibid.* (1900), 21, 273.
[7] Albert. *This Journ., Sec. B* (1908), 3, 345.

along the junction of the lobes. Occasionally there is a slight increase of the pleural fluid.

A cut section shows a pinkish-gray surface, which may or may not exude some blood. Air can be expressed from all portions of the lung and usually also a slight amount of œdematous fluid. The posterior part of the lung is of a darker color and is heavier than the anterior and more fluid can be expressed from it than from the anterior part. The bronchi do not appear to be hyperæmic, but contain more or less frothy material and mucus. Sometimes this can be expressed from the smaller bronchi.

Spleen.—This organ may be very hyperæmic and show slight increase of splenic tissue, partially obliterating the normal markings.

Kidneys.—The kidneys are of a reddish-gray color, fœtal lobulations are prominent. A cut section is very moist and a considerable amount of blood oozes from it. Striations of the cortex are plainly seen. Except for congestion, the kidneys, in the greater number of cases, present a normal appearance. Occasionally a slight degree of albuminous degeneration, or a few subcapsular hæmorrhages occur. The adrenals show congestion.

Liver.—The liver is dark reddish-brown in color and firm. Section shows congestion and rarely a slight "nutmeg" appearance is seen. The lobulations are usually clearly defined. Here, also, we may find some albuminous degeneration.

Stomach.—The stomach nearly always contains some curdled milk and mucus. The mucosa is smooth and anæmic. No rice or other artificial food was found in the stomachs of any of the cases; sometimes there is a trace of fæcal material present.

Intestines.—They are normal in appearance. The intestinal contents are semiliquid, apparently digested, and have a yellow color. The mesenteric glands may be slightly enlarged and soft.

Urinary bladder.—It may or may not contain urine.

Throat organs.—Except for some froth and mucus present in the larynx and trachea, these organs are normal.

Thymus.—The thymus is usually prominent and full. Some milky fluid can almost always be expressed from the cut surface.

Meninges and brain.—The meninges are congested and œdematous, and there is usually an increase of the cerebro-spinal fluid. The brain substance may be of normal consistency, or soft and very moist.

Many of the bodies were not subjected to necropsy until twenty-four or thirty-six hours after death, because of our inability to transport them to the morgue at a sufficiently early time, and in this warm climate decomposition very rapidly sets in. However, in the above description of the gross pathology three points are prominent: First, the dilated and hypertrophied right heart; second, the congestion of all internal viscera; third, the anasarca. The two last conditions naturally follow the first, and we turn to the lungs for a possible explanation. Microscopically they present a varied condition, being in part emphysematous and the more dependent portions being heavily congested, with the alveoli filled with epithelial cells, leucocytes, and granular œdematous material. In some places small hæmorrhages have occurred and the red cells are found in the alveoli and tissues.

In this paper we do not attempt to give the etiology of the condition. A microbic origin for it is not excluded, as we have not been able to enter upon this phase of the investigation because of the length of time elapsing between the death of the patient and the time of necropsy, and the press of other work; but if the condition is of microbic origin, its manifestation is entirely different from that produced by other organisms that we know. There is no inflammatory focus in the lungs or elsewhere.

Clinically, the cases were not seen by either of us, and all the information we have was obtained by the medical inspectors and is contained in the tables. The inspectors give the history that the patient is sick but a few hours, or, at most, but a day or two. The parents, however, do not recognize that the child is ill until its condition is very serious, or until it is almost dead. The probability is that it has been ailing for some time, possibly since its birth.

Nearly all of the infants examined were under 2 months of age, and but few were above 2½ months old. Almost without exception they were breast fed, and in no case was rice or other artificial food found in the stomach. The mothers in nearly all instances exhibited some symptoms of beriberi. Many of them give the history of having two, three, or even five or more children die from similar symptoms. The disease is well known among the native poor, who call it *"taon"* or *"suba"* (propably the latter is the more common name), and who dread it very much.

In a careful analysis of the pathologic findings by necropsy in a series of 219 infants dying under 1 year of age, we found the above-described condition, which, for want of a better term, we have designated as "moist beriberi," to be present in 124 cases. The following summary gives an analysis of all of those cases.

Table showing results of necropsies on 219 infants 1 year of age or under.

Beriberi	124
Cholera	33
Pneumonia	18
Meningitis	6
Enterocolitis	6
Other diseases	20
Undetermined [a]	12
Total	219

[a] In some of the undetermined cases the findings were obscured by post-mortem decomposition; other bodies were greatly emaciated and death seemed to be due to a lack of assimilation of food.

This table includes all the cases discussed in the preceding investigations under the headings meningitis, enteritis, convulsions, bronchitis and beriberi, excepting a few cases of meningitis over 1 year of age, which were excluded. The total was increased by including some necropsies made during March and April, after the other investigations were concluded.

The table indicates that "beriberi" (?) is the largest factor in the infant mortality of Manila, and the existence of this factor accounts in great measure for Manila's excess in infant mortality over that of other countries, as shown by Table III. Cholera appears because the investigations were begun during a mild epidemic of that disease in Manila. Tubercular meningitis was found twice and tuberculosis of the lungs once. This does not indicate the measure of the prevalence of tuberculosis among Filipino infants, as deaths from this disease were not investigated.

GENERAL CONSIDERATIONS.

The infant mortality of Manila presents a striking contrast to that of other cities. The deaths of breast-fed children constitute 73.74 per cent of the total infant mortality; furthermore, 87 per cent of infants dying of beriberi and convulsions in Manila are breast fed. The following table shows the figures as compiled in the Bureau of Health for the year ending February 25, 1910.

TABLE XII.—*Showing per cent of deaths among breast-fed infants under 1 year, by disease.*

Diseases.	Number of certificates stating how fed.	Number breast-fed.	Per cent of breast fed to total reported on.
Beriberi	473	417	88.1 +
Convulsions	872	763	87.5
Acute bronchitis	615	469	76.2 +
Congenital debility	381	218	57.2 +
Meningitis	134	75	56 —
Enteric disease	201	70	34.8 +
Other diseases	722	494	68.4 +
Total	3,398	2,506	73.74 +

In Munich, of 4,000 dead infants during 1903, 83 per cent were artificially fed;[9] in Berlin, in a period of five years, only 9 per cent of the infant mortality occurred in breast-fed babies.[10]

[9] Wile. *Pediatrics* (1909), 21, 203.
[10] Graham. *Journ. Am. Med. Assoc.* (1908), 51, 1045.

TABLE XIII.—*Showing per cent of total infant mortality which occurs in breast-fed and artificially fed infants in different cities.*

City.	Breast-fed.	Artificially fed.
	Per cent.	*Per cent.*
Berlin	9	91
Munich	17	83
Manila	73.74	26.26

Tugendreich [11] states that in 64 families with 388 breast-fed children there had been 77 deaths, a mortality of 19.8 per cent. On the other hand, he found that 33 families with 229 bottle-fed children had 99 deaths, a mortality of 43.2 per cent. Twenty-four of the 64 families with 109 exclusively breast-fed children escaped without a single death, while not one of the 33 families with bottle-fed children escaped without the loss of at least one child. In other countries the mortality among breast-fed infants is very low.

In Germany, France, or the United States a breast-fed infant means a healthy infant in 90 per cent of the cases, because the mothers in those countries are usually healthy and well nourished. In the Philippines the mortality is greatest among breast-fed children, possibly because of the poor quality of the mother's milk. The latter is probably deleterious by reason of what it lacks rather than because of any harmful constituent. The average Filipino mother is in poor physical condition, many of them are beriberic and subsist upon a diet favorable to beriberi. It seems probable that there is an intimate relation between beriberi of infants and a mother's milk poor in quality and lacking certain necessary elements which are not included in the mother's dietary. At first glance it might seem advisable to supplant breast feeding by artificial, but under existing conditions this would be a blunder. The children saved from beriberi would be sacrificed to enteric diseases. That small part of our infant population which is artificially fed furnishes 65 per cent of the deaths from enteric diseases, and the breast-fed, much the larger part of the population, furnishes but 35 per cent of the infant mortality from this cause; so that even in Manila, breast-feeding of infants exerts a deterrent influence upon the mortality from gastrointestinal diseases. A possible solution of the problem lies in improving the quality of the mother's milk and encouraging the continuance of the custom of breast-feeding so general among the Filipino poor. The improvement of the physical condition of the Filipino mother and of the quality of her milk is an economic question. Her condition is the result of poverty and therefore insufficient and unsuitable food, especially during the periods of pregnancy and lactation.

[11] *Arch. f. Kinderheilk.* (1908), 48, 390.

However, it must be remembered that the etiology of this disease is unknown. Clinically, the cases must be observed by competent men under favorable conditions where a complete clinical record can be made. The sick ones should be taken to a hospital where they can be studied carefully and all laboratory and biologic tests applied. The careful examination of all body fluids and excreta, biologically and chemically, is imperative. Further, the possibility of an ultramicroscopic organism must not be overlooked, neither should the internal secretions be forgotten. At the same time, a thorough chemical and biologic examination of the mother's milk is essential. At death a quick necropsy with complete biologic examination must follow. This would also give material for thorough histologic study, which has been impossible so far because of post-mortem changes.

The writers wish to extend their thanks to Dr. H. Winsor, of St. Paul's Hospital, for performing some of the autopsies.

ILLUSTRATIONS.

PLATE I.

FIGS. 1–3. Hearts of infants dead of beriberi. The increase in size of the right ventricle is apparent.

FIG. 4. Heart of an infant dead of beriberi sectioned to show the large size of the right ventricle and the relatively small size of the left.

TABLE IX.—*Cases of alleged bronchitis or broncho-pneumonia.*

Clinic.	Clinical data.									Pathologic findings.										
	Age.			Fever.	Cough.	Dyspnœa.	Cyanosis.	Diarrhœa.	Convulsions.	Beriberic symptoms in mother.	Feeding.	External appearance of choleræ.	Subcutaneous tissues.	Peritoneum.	Heart.	Lungs.	Spleen.	Kidneys.	Liver.	Intestines.	Meninges and brain.	Anatomic diagnosis.	Bacteriologic diagnosis.
	y.	m.	d.																				
1	—	1	—	Yes	Yes	Yes	Yes	No				No	Moist	Moist	Right dilated and hypertrophied.	Œdema	Normal	Congestion	Congestion	Normal	Congestion	Beriberi.
2	—	4	—	Slight	Yes	Yes	Yes	No			Breast	No	do	Ascites	do	Pulmonary œdema	Congestion	do	do	do	Congestion, œdema	do.
3	—	1	7	No	Yes	Yes	Yes	No	Yes	Yes	do	No	Firm	Moist	do	Pneumonia		Albuminous degeneration	Albuminous degeneration	do		do.
4	—	—	15	Yes		Yes	Yes	No	Yes	No	do	No	Firm	Normal	Normal	Pneumonia		Postmortem decomposition				Bronchopneumonia.
5	—	—	15	Yes	Yes	Yes	Yes	No	Yes	No	do	No	Œdema	Ascites	Right dilated and hypertrophied.	Pulmonary œdema	Normal		Congestion	Normal	Congestion, œdema	Beriberi.
6	—	5	15	Yes	Yes	Yes	Yes	No	Yes	Yes	do	No	do	do	do	Pneumonia		Postmortem decomposition				do.
7	—	11	—	Yes	Yes	Yes	Yes	No		No	Mixed	No	Firm	do	Normal	Œdema	Normal	Congestion	Albuminous degeneration	Normal	Meningitis	Suppurative meningitis.
8	1	1	—	Yes	Yes	Yes	Yes	Yes	Yes	No	Artificial	No	do	Normal	do	Pneumonia	do	Albuminous degeneration	do	do	Congestion, œdema	Lobar pneumonia.
9	—	10	—	Yes	Yes	Yes	Yes	No	No		do	No	do	do	do			Postmortem decomposition				Acute nephritis.
10	—	6	7	Yes	Yes	Yes		No		No	do	No	do	do	do	Œdema	Normal	Nephritis	Normal	Normal	Normal	Beriberi.
11	—	7	8	Yes	Yes	Yes	Yes	No	No	No	Breast	No	Œdema	Ascites	Right dilated and hypertrophied.	Pulmonary œdema	Congestion	Congestion	Congestion	do	Meningitis	Suppurative meningitis.
12	—	4	3	Yes	No	Yes		No	Yes	No	do	No	Firm	Normal	Normal	Œdema	Normal	Albuminous degeneration	do	do		Chronic colitis.
13	—	5	15	Yes	Yes	Yes	Yes	No	No	No	do	No	Dry	do	do	Postmortem decomposition					Colitis	
14	—	8	—	No	Yes	Yes	Yes	No	No	No	do	No			Right dilated and hypertrophied.							Beriberi.
15	—	2	24	Yes	Yes	Yes				No	do	No	Moist	Ascites	do	Pulmonary œdema	Normal	Congestion	Congestion	Normal		do.
16	—	1	10	No		Yes	Yes			No	do	No	do	do	do	Postmortem decomposition						do.
17	—	2	25	Yes	Yes	Yes		No	No	Yes	do	No	Œdema	do	do	Pulmonary œdema	Normal	Congestion	Congestion	Normal		do.
18	—	2	18	Yes	Yes	Yes		No	No	No	do	No	Moist	Moist	Normal	Œdema	do	do	do	do	Congestion	Cysticercus cellulosæ.
19	—	3	4	Yes	Yes	Yes		No	No	No	do	No	Firm	do	Right dilated and hypertrophied.	Pneumonia	Congested	Albuminous degeneration	Albuminous degeneration	do		Lobar pneumonia.
20	2	5	—	Yes	Yes	Yes		No	No	No	Mixed	No	do	do	Normal	Congestion, bronchitis	Normal	Congestion	Congestion	do	Congestion	Acute tonsilitis bronchitis.
21	—	2	—	Yes	Yes	Yes		No	No	No	Breast	No	do	do	Right dilated and hypertrophied.	do	Congested	do	do	do		Beriberi.
22	—	1	21	Yes	Yes	Yes		No	No	No	do	No	do	Ascites	Normal	do	do	Nephritis	do	do		Nephritis.
23	—	2	15	Yes	Yes			No	No	No	Artificial	No	Œdema	do	Right dilated and hypertrophied.	Congestion	do	Congestion	do	do	Congestion	Beriberi.
24	—	8	—	Yes	Yes	Yes		No	No	No	Breast	No	Firm	Moist	Normal	Pneumonia	Normal	Normal	Normal	do		Bronchopneumonia.
25	—	2	—	Slight	Yes	Yes		No	No	Yes	do	No	Œdema	Ascites	Right dilated and hypertrophied.	Congestion, œdema	Congestion	Congestion	Congestion	do	Congestion, œdema	Beriberi.
26	—	3	—	No	No	Yes	Yes	No	No	No	do	No	do	Moist	do	do	do	do	Congestion	do	Œdema	do.
27	—	4	—	Yes	Yes	Yes	Yes	No	Yes	No	do	No	Firm	do	Normal	Pneumonia	Normal	Albuminous degeneration	Albuminous degeneration	do		Broncho-pneumonia.

[a] Greatly emaciated infant; death probably due to lack of assimilation of food.

TABLE X.—*Cases of alleged infantile beriberi.*

Case.	Clinical data.[a]										Pathologic findings.									
	Age.			Beriberic mother.	Feeding.	Convulsions or convulsive symptoms.	Fever.	Urine.	Dyspnœa and cyanosis.	Vomiting.	Diarrhœa.	External appearance of cholera.	Subcutaneous tissues.	Peritoneum.	Heart.	Lungs.	Spleen	Kidneys.	Liver.	Intestines.	Meninges and brain.	Anatomic diagnosis.	Bacteriologic diagnosis.
	y.	m.	d.																			
1	—	1	21	Yes	Breast	No	Yes	Scanty	Yes	No	No	Yes	Dry	Dry	Normal	Œdema	Congestion	Albuminous degeneration	Albuminous degeneration	Inflamed	Congestion	Cholera	Negative.
2	—	5	26	Yes	do	No	Yes	Diminished	Yes	No	No	No	Moist	Ascites	Right dilated and hypertrophied.	Congestion	Normal	Congestion	do	Normal	do	Beriberi	Do.
3	—	1	2	Yes	do	No	Yes	Anuria	Yes	No	No	No	Œdema	Moist	do	Congestion, œdema	Congestion	Albuminous degeneration	do	Inflamed	do	Cholera	Do.
4	—	1	—	No	do	No			Yes	No	No	Yes	Dry	Dry	Normal	do	do	do	do	Normal	do	Beriberi	Do.
5	—	2	12	Yes	do	No	Slight		Yes	No	Soft yellow stools	No	Moist	Moist	Right dilated and hypertrophied.	do	do	do	do	do	do	do	No report.
6	—	3	—	Yes	do	No		Suppressed	Yes	Yes	do	No	do	do	do	do	do	Congestion	do	do	do	do	Negative.
7	—	2	—		do	No	Yes		Yes	Yes	Yellow	No	do	Ascites	do	do	do	do	do	do	do	Broncho-pneumonia	Do.
8	—	3	—	Yes	do	No	No		Yes	Yes	No	No	do	do	Normal	Broncho-pneumonia	Normal	Acute nephritis	do	do	do	Enterocolitis	
9	1	—	—	No	Artificial	No	No	Diminished	Yes	Yes	No	Yes	do	do	do	Congestion	do	Congestion	Congestion	Inflamed	Congestion	Broncho-pneumonia	Negative.
10	—	3	—	Yes	Breast	No	No		Yes	Yes	No	No	do	do	do	Broncho-pneumonia	Congestion	do	Albuminous degeneration	Normal	do	Beriberi	Do.
11	—	5	—	No	do	No	No		Yes	Yes	No	No	do	Moist	Right dilated and hypertrophied.	Congestion, œdema	do	Albuminous degeneration	do	do	do	do	
12	—	1	17			No	No	Diminished	Yes	No	No	No	do	do	do	Congestion	Normal	Congestion	do	do	do	do	Negative.
13	—	2	—	Yes	Breast	No	No	do	Yes	No	No	No	do	do	do	Congestion, œdema	Congestion	do	Albuminous degeneration	do	do	do	Do.
14	—	1	—	Yes	do	No	No	do	Yes	No	No	No	do	Ascites	do	do	Normal	do	Albuminous degeneration	do	Normal	do	Do.
15	—	2	—	Yes	do	No	No	Suppressed	Yes	No	No	No	do	Moist	do	Congestion	do	Albuminous degeneration	Congestion	do	do	do	
16	—	1	25	Yes	do	No	No	Scanty	Yes	No	No	No	do	do	do	do	do	Congestion	do	do	do	do	Negative.
17	—	2	19	Yes	do	No	No	Anuria	Yes	No	No	No	do	do	do	Congestion, œdema	do	Albuminous degeneration	do	do	Œdema	do	Do.
18	—	4	—	Yes	do	No	No		Yes	No	No	No	do	do	do	do	Congestion	Congestion	do	do	do	do	Do.
19	—	6	—	No clinical history obtainable								No	do	do	do	do	Normal	do	Congestion	do	Congestion, œdema	do	Do.
20	—	1	20			No	No	Diminished	Yes	No	No	No	do	Ascites	do	do	do	do	do	do	Normal	do	Do.
21	—	1	9	No	Breast	No	No	do	No	No	No	Yes	Post-mortem decomposition									Cholera (?)	Do.
22	—	1	21	Yes	do	No	No	Anuria	Yes	Yes	No	No	Moist	Moist	Right dilated and hypertrophied.	do	Congestion	Congestion	Congestion	Normal	Congestion	Beriberi	Do.
23	—	2	—	No	do	No	No	Scanty	Slight	No	No	No	do	Ascites	do	do	do	do	do	do	do	do	No report.
24	—	1	3	Yes	do	No	No	do	do	No	No	No	do	do	do	do	Normal	do	do	do	Œdema	do	Negative.
25	—	1	17	Deaf mute.	do	No	No	Anuria	do	No	No	No	do	do	do	Congestion, œdema	Congestion	do	do	do	do	do	Do.
26	—	1	19	Yes	do	No	Slight	Diminished	do	Yes	No	No	do	do	do	Congestion	do	do	do	do	do	do	
27	—	1	27	Yes	do	No	No	Anuria	do	No	No	No	do	Moist	do	Congestion, œdema	Normal	do	do	do	Normal	do	
28	—	1	7	Yes	do	No	No		do	No	No	No	do	do	do	do	do	do	do	do	Œdema	do	
29	—	3	—	No	do	No	No		do	No	No	No	do	do	do	do	do	Congestion, albuminous degeneration	do	do	Normal	do	
30	—	5	—	Yes	do	No	No	Anuria	do	No	No	No	Œdema	Ascites	do	do	Congestion	Congestion	do	do	Congestion	do	
31	—	2	5	Yes	do	No	No	do	do	No	No	No	do	do	do	do	do	do	do	do	do	do	
32	—	1	13		do	No	No		do	No	No	No	do	Moist	do	do	do	do	do	do	Congestion, œdema	do	
33	—	2	—	Yes	do	No	No		do	No	No	No	do	do	do	do	do	do	do	do		Undetermined[b]	
34	—	2	—	Yes	Mixed	Yes	No		Yes	Yes	Yes	Yes	Post-mortem decomposition									Beriberi	
35	—	2	23	Yes	Breast	No	No		Yes	Yes		No	Moist	Moist	Right dilated and hypertrophied.	Congestion, œdema	Congestion	Congestion	Congestion	Normal		do	
36	—	2	—	Yes	do	No	No	Diminished	Yes			No	Œdema	Ascites	do	do	do	Congestion, albuminous degeneration	do	do		Undetermined[b]	
37	—	1	20	Yes	do	No	No		Yes			No	Post-mortem decomposition									Beriberi	
38	—	1	6	Yes	do	No	No	Diminished	Yes	No	No	No	Moist	Ascites	Right dilated and hypertrophied.	do	Normal	Congestion	Congestion	Normal		do	
39	—	1	10	Yes	do	Yes	No	Scanty	Yes	No	No	No	Œdema	Moist	do	do	Congestion	do	do	do		do	
40	—	3	—	Yes	do	No	No	Anuria	Yes	No	No		Post-mortem decomposition									do	
41	—	2	—	No	do	No	No	do	Yes	No	No	No	Œdema	Ascites	Right dilated and hypertrophied.	Congestion, œdema	Congestion	Congestion, albuminous degeneration	Congestion	Normal	Congestion, œdema	do	
42	—	1	6	Yes	do	No	No	do	Yes	No	No	No	do	do	do	do	do	do	do	do	do	do	
43	—	2	10	Yes	do	No	No	do	Yes	No	No	No	do	do	do	Congestion	do	do	Congestion	do	Œdema	do	
44	—	1	27		do	Yes	No		Yes	No	No	No	Moist	do	do	Congestion, œdema	Normal	Congestion	Congestion	do		do	
45	—	1	7		do	No	No	Diminished	Yes	No	No	No	do	Moist	do	do	Congestion	do	do	do		do	
46	—	2	29	Yes	do	No	No	do	Yes	No	No	No	Œdema	Ascites	do	do	do	do	do	do		do	
47	—	10	—	No	do	Yes	Yes		Yes	Yes	No	No	do	Moist	do	do	Normal	do	do	do		do	
48	—	1	29	No clinical history obtainable								No	Moist	do	do	do	Congestion	do	do	do		do	
49	—	4	13	Yes	Breast	No	Slight	Diminished	Yes	Yes		No	do	Ascites	do	Pneumonia	do	do	Congestion, albuminous degeneration	do	Œdema	Pneumonia	
50	—	4	2	Yes	do	No	No	do	Yes	No	No	No	do	do	do	Congestion, œdema	do	do	do	do	Congestion	Beriberi	

[a] In a great many cases the clinical observers noted changes in the voice, varying from weakness and hoarseness to complete aphonia.

[b] Post-mortem decomposition.

TABLE VI.—*Cases of alleged meningitis.*

Case.	Clinical data.									Pathologic findings.									
	Age.			Convulsions or convulsive symptoms.	Rigidity of neck.	Strabismus.	Fever.	Collapse.	Vomiting.	Rice water diarrhœa.	External appearance of cholera.[a]	Subcutaneous tissues.	Peritoneum.	Heart.	Lungs.	Spleen.	Kidneys.	Liver.	Intestines.	Meninges and brain.	Anatomic diagnosis.	Bacteriologic diagnosis.
	y.	m.	d.																		
1	3	—	—	Yes	No	No	Slight	No	No	No	Yes	Dry	Dry	Normal	Congestion	Normal	Nephritis	Albuminous degeneration	Slight congestion	No inflammation	Cholera	Cholera.
2	2	4	—	No	Yes	Yes	Yes	No	No	Facculent diarrhœa	Yes	do	do	Right, dilated	Congestion and œdema	do	Albuminous degeneration	do	Congestion	Congestion	do	Do.
3	—	10	—	Yes	No	Yes	Yes	No	Nausea	Yellow diarrhœa	Yes	do	do	do	do	do	Nephritis	do	Inflamed	Congestion, œdema	do	Do.
4	1	—	—	Yes	No	Yes	Yes	No	No	No	Yes	do	do	do	do	do	do	Congestion	do	do	do	Negative.
5	1	—	—	Yes	No	Yes	Yes	No	No	Constipation	Yes	do	do	do	Congestion	do	do	do	do	do	do	Cholera.
6	3	—	—	No	No	Yes	High	No	No	No	Yes	do	do	do	do	do	Congestion	Albuminous degeneration	do	Congestion	do	Do.
7	3	—	—	Yes	Yes	No	No	Cold extremities	Yes	Yes	Yes	do	do	do	do	do	Nephritis	Congestion	do	Congestion, œdema	do	Negative.
8	—	6	—	No	No	Slight	Yes	No	No	No	Yes	do	do	do	do	do	Congestion	do	Normal	Suppurative congestion	Suppurative meningitis	Do.
9	3	—	—	Yes	Yes	No	Yes	No	No	No	No	Moist	Moist	Normal	do	do	Nephritis	Normal	do	Normal	Undetermined[b]	Do.
10	—	10	—	No	No	No	Slight	No	No	Facculent diarrhœa	No	do	do	do	Normal	do	Normal	do	do	Congestion	Pseudo-lobar pneumonia[b]	No report.
11	—	5	—	Yes	Yes	No	Yes	No	No	Yellow-diarrhœa	No	do	do	do	Pneumonia	Congestion	Nephritis	do	do	Congestion, œdema	Undetermined	Negative.
12	—	3	—	Yes	No	No	Yes	No	Yes	do	No	Dry	Dry	Soft	Congestion	Normal	Normal	do	Inflamed	Congestion	Cholera	Cholera.
13	2	—	—	Yes	No	Yes	Yes	No	No	No	Yes	do	do	Right, dilated	do	do	Nephritis	Congestion	do	No inflammation	do	Negative.
14	3	6	—	Yes	No	No	High	No	No	Yellow, liquid stools	Yes	do	do	do	Congestion and œdema	do	Congestion	Albuminous degeneration	do	Congestion	do	Cholera.
15	—	8	—	No	Slight	Yes	Slight	No	Nausea	No	Yes	do	do	do	Congestion	do	Albuminous degeneration	do	do	Congestion	do	Do.
16	1	1	—	Yes	No	No	High	No	do	Greenish yellow stools	Yes	do	do	do	do	do	Congestion	do	do	do	Undetermined[c]	Do.
17	—	8	—	Yes	No	No	Slight	No	No	Greenish-yellow liquid stools.	Yes	Post-mortem decomposition										
18	5	7	—	Slight	No	No	Yes	No	No	No	Yes	Dry	Dry	Right, dilated	Congestion	Normal	Albuminous degeneration	Albuminous degeneration	Inflamed	No inflammation	Cholera	Do.
19	—	4	—	Yes	Yes	Yes	Yes	No	No	No	Yes	do	do	do	do	do	do	do	do	Congestion	do	Do.
20	8	—	—	Yes	No	No	Yes	No	No	No	Yes	do	do	do	do	do	do	do	do	No inflammation	do	Do.
21	1	—	—	No	No	No	Slight	No	No	No	Yes	do	do	do	do	do	do	do	Congestion	Congestion	do	Do.
22	—	11	—	No	No	No	Yes	No	No	No	No	Slight œdema	Ascites	Right, dilated and hypertrophied	do	Congestion	Congestion	do	Normal	Normal	Beriberi	Negative.
23	5	—	—	Yes	No	Yes	Yes	No	No	No	No	Moist	do	do	Congestion and œdema	do	Congestion	do	do	Congestion	do	Do.
24	—	8	—	Yes	No	Ptosis	Yes	No	Yes	No	No	do	Moist	do	Congestion	Normal	Albuminous degeneration	do	Slight congestion	do	do	Do.
25	—	2	—	Yes	Yes	Yes	Yes	No	No	No	No	do	do	do	Broncho-pneumonia	Congestion	do	do	do	do	do	Do.
26	—	4	—	Yes	Yes	Yes	High	No	No	No	No	Firm	Normal	Dilated and hypertrophied	Empyema	do	Congestion, albuminous degeneration	do	Normal	Suppurative meningitis	Meningitis	Do.
27	—	3	—	No	No	Yes	High	No	No	No	No	do	Moist	do	Congestion	do	Albuminous degeneration	do	Congestion	Congestion	Beriberi	Do.
28	—	11	—	Yes	Yes	Yes	Yes	No	Nausea	No	Yes	Dry	Dry	Right, dilated	Congestion and œdema	Normal	Congestion	Congestion	Inflamed	Congestion, œdema	Cholera	No report.
29	—	10	—	Yes	No	Yes	Yes	No	Yes	Soft, yellow stools	No	Moist	Ascites	Right, dilated and hypertrophied	do	Congestion	Albuminous degeneration	Albuminous degeneration	do	do	Beriberi	Negative.
30	—	7	—	Yes	No	No	Yes	No	No	do	No	Firm	Normal	Normal	Congestion	do	Normal	Congestion	Normal	Slight congestion	Empyema	Do.
31	—	8	—	Yes	Yes	Yes	Yes	No	Yes	No	No	Moist	Ascites	Right, dilated and hypertrophied	do	do	Congestion	do	do	Congestion, œdema	Beriberi	Do.
32	2		s.	Yes	Yes	Yes	Yes	No	Yes	No	Yes	Dry	Dry	Right, dilated	do	Normal	Albuminous degeneration	Albuminous degeneration	Inflamed	Congestion	Cholera	No report.
33					No		Yes	No	No	No	No	Moist	Normal		Pneumonia	do	Congestion	Congestion	Congestion	do	Pneumonia	
34					Yes		Yes	No	Yes	Yellow	No	Firm	Ascites	Right, dilated and hypertrophied	Congestion œdema	Congestion	Congestion	Congestion	Normal	Congestion, œdema	Beriberi	
35					No			No	Nausea	No	No	Moist	do		Congestion	Normal	Congestion, albuminous degeneration	Congestion, albuminous degeneration	Inflamed	Normal	do	
					Yes				No	No	No	do	Ascites		Congestion and œdema	Congestion	do	do	do	Congestion, œdema	do	
					Yes				No	No	No	Dry	do	Normal	Congestion	Normal	Congestion	do	Inflamed	Normal	Acute colitis	

[a] By [illegible] have reference to a picture presenting the following: A haggard drawn expression of the face with sunken, half open eyes. The finger nails are bluish in color, the skin of the fingers is shriveled and wrinkled, and the skin covering the body is drawn and tight, as a rule.

[b] Greatly [illegible] to nonassimilation of food.

[c] Post-mortem [illegible]

TABLE VII.—*Cases of alleged enteritis and other diarrhœas.*

Case.	Clinical data.									Pathologic findings.										
	Age.			Fever.	Vomiting.	Collapse.	Urine.	Character of diarrhœa.	Convulsions or convulsive symptoms.	Dyspnœa and cyanosis.	Clinical diagnosis.	External appearance of cholera.	Subcutaneous tissues.	Peritoneum.	Heart.	Lungs.	Spleen.	Kidneys.	Liver.	Intestines.	Meninges and brain.	Anatomic diagnosis.	Bacteriologic diagnosis.
	y.	m.	d.																			
1	5	—	—	Yes	No	No	Abundant	Fœtid, dark	No	Yes	Typhoid fever	Yes	Dry	Dry	Normal	Congestion	Normal	Congestion	Albuminous degeneration	Inflamed	Congestion	Cholera	Cholera.
2	4	—	—	Yes	No	No		Mucus and blood	No	Yes	Acute dysentery	Yes	do	Sticky exudate	do	do	do	Albuminous degeneration	do	do	Normal	do	Negative.
3	—	1	—	Yes	Yes	No	Scanty	Greenish, pasty	No	No	Acute enteritis	Yes	Moist	do	Right dilated	Congestion	do	Congestion	do	do	do	do	Cholera.
4	—	1	—	No	Once	Yes	Sufficient	Yellow	No	Yes	Acute gastroenteritis	Yes	Dry	Dry	do	do	do	do	do	do	Congestion	do	Do.
5	—	3	—	No history obtainable							Acute enteritis	No	Moist	Ascites	Right dilated and hypertrophied			Congestion			do	Beriberi	Negative.
6	—	6	—	do							do	Yes	Dry	Dry	Normal	do	Congestion	do	Congestion	Inflamed	Normal	Cholera	Do.
7	1	—	—	Yes	Yes	No		Greenish-yellow	No	Slight cyanosis.	Chronic gastroenteritis	Yes	do	do	Right dilated	do	Normal	Albuminous degeneration	Albuminous degeneration	do	Congestion	do	Do.
8	6	—	—	Yes	No	No		Mucus and blood	No	No	Acute dysentery	No necropsy; specimen of intestinal contents taken after death											Cholera.
9	1	6	—	Yes	No	No		Greenish stools	No	Slight	Acute gastroenteritis	do											Do.
10	2	—	—	Yes	Yes	No	Scanty	Light yellow	No	No	Acute enteritis	Case recovered; diagnosis from stool specimen											Do.
11	2	—	—	Yes	No	No	Normal	Fœculent	No	No	Acute gastroenteritis	do											Do.
12	2	—	—	Yes	Yes	No		do	No	No	do	No necropsy; specimen of intestinal contents taken after death											Do.
13	3	—	—	Slight	No	No	Normal	do	No	No	Chronic tubercular enteritis	do											Do.
14	3	6	—	Yes	No	No		Mucus and blood	No	No	Acute dysentery	do											Do.
15	1	—	—	No clinical history obtainable							Acute enteritis	No	Moist	Moist	Normal	Pneumonia	Normal	Albuminous degeneration	Albuminous degeneration	Normal	Normal	Lobar pneumonia	No report.
16	1	9	—	do							Acute gastroenteritis	Yes	Dry	Dry	Right dilated	Congestion	do	Congestion	do	Inflamed	do	Cholera	Cholera.
17	—	5	—	do							Chronic gastritis	No	Moist	Moist	Right dilated and hypertrophied	do	do	do	Normal	Normal	Congestion	Beriberi	Negative.
18	—	—	27	do							Acute gastroenteritis	No	do	do	Right dilated	Pneumonia	Congestion	do	do	do	do	Pneumonia	No report.
19	—	6	—	Yes	Yes	No			Yes	Yes	do	Yes	Dry	Dry	do	Congestion	do	Congestion, albuminous degeneration	Albuminous degeneration	Inflamed	do	Cholera	
20	1	—	—	Yes					Yes		do	No	do	Moist	Normal	Congestion and œdema	do	Congestion	do	do	Normal	Acute enteritis	
21	—	4	—					Mucus			Undetermined	No	do	Dry	do	Œdema	do	Albuminous degeneration	Congestion	do		Acute enterocolitis	
22	—	10	—	Yes	Yes		Plentiful	Dysentery			Chronic gastritis	Yes	do	do	do	Normal	Normal	do	Albuminous degeneration	do	Congestion, œdema	do	

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TABLE VIII.—*Cases of alleged infanticide examinations.*

Case.	Clinical data.													Pathologic findings.						
	Age.			Convulsions or convulsive symptoms.	Fever.	Collapse.	Vomiting.	Diarrhea.	Dyspnea and cyanosis.	Urine.	External appearance of cholera.	Subcutaneous tissues.	Peritoneum.	Feces.	Spleen.	Kidneys.	Liver.	Intestines.	Condition and bulk.	Atheromatous diathesis.	Physiologic changes.
	y.	m.	d.																		
1	3	—	—	Yes	Yes	No	No	Yes	No		Yes	Dry	Dry	Normal	Atrophied	Normal	Albuminous degeneration	Albuminous degeneration	Inflamed	Normal	Cholera.
2	—	1	—	Yes	No	No	No	No	Yes	Amount normal	No	Moist	Moist	Bright blood and hyperemia	do	Congestion	Congestion	Congestion	Normal	Normal	Do.
3	1	—	—	Yes	No	Cold extremities	Yes	Yes	Slight	Do	Yes	do	Slight ascites	Normal	Congested	do	Congestion	Congestion	Normal	Normal	No cholera.
4	—	2	—	Yes	No	No	No	No	Yes		No	do	do	Bright blood and hyperemia	do	Albuminous degeneration	Albuminous degeneration	Normal	Normal	Normal	Do.
5				No history obtainable.							No	do	do	do	do	Normal	do	do	Normal	Do.	Do.
6	—	1	18	Yes	No	No	No	Yellow	Yes	Diminished	No	do	Moist	do	do	Nephritis	do	do	Normal	Do.	Do.
7	—	1	19	Yes	No	No	No	No	Yes	Do	No	do	Slight ascites	do	Normal	Congestion	do	do	Normal	Do.	Do.
8	—	2	—	Yes	No	No	No	No	Yes		No	do	Ascites	do	do	Congestion	do	do	do	Do.	Do.
9	—	2	16	Yes	No	No	No	Yellow	No	Amount normal	Yes	Postmortem decomposition								Thrombosis	Do.
10	—	—	27	Slight	No	No	No	No	No		No	Moist	Moist	Bright blood and hyperemia	Congested	Congestion	Congestion	Normal	Normal	Bronchitis	Do.
11	—	3	32	Yes	Yes	No	No	No	Yes	Diminished	No	do	Slight ascites	do	do	do	do	do	do	do	Do.
12	—	1	15	Slight	No	No	No	No	No		No	do	Moist	do	do	do	do	do	do	do	Do.
13	—	1	8	No	Yes	No	No	No	No		No	do	Slight ascites	do	Normal	Nephritis	Albuminous degeneration	do	Normal	do	Do.
14	—	2	—	Yes	No	No	Yes	Greenish liquid	No	Amount normal	No	do	do	do	Congested	Normal	Congestion	do	Normal	do	Do.
15	—	2	—	Yes	No	No	No	No	Yes		No	do	do	do	do	Albuminous degeneration	do	Slight congestion	Congestion	do	
16	—	2	—	No	No	No	No	No	Yes	Diminished	No	do	do	do	Normal	Congestion	Congestion	Normal	Normal	do	
17	—	2	—	No	No	No	No	Greenish liquid	Yes	do	No	do	Moist	do	do	Albuminous degeneration	do	do	do	do	Do.
18	—	1	9	Yes	No	No	No	Yellow	Yes	Suppressed	No	do	do	do	do	Normal	Albuminous degeneration	do	Congested	do	Do.
19	—	7	—	Yes	No	No	No	No	Yes	Diminished	No	do	do	Purulent (colorless)	do	Congestion	Albuminous degeneration	do	Normal	Congestion	Do.
20	—	6	22	Yes	No	No	No	No	No		No	do	Slight ascites	Bright blood and hyperemia	do	Normal	Congestion	Normal	Normal	Do.	Do.
21	—	1	25	Yes	No	No	No	Purulent	Yes	Amount normal	No	do	Moist	do	do	Congestion	do	Albuminous degeneration	do	do	Do.
22	—	6	15	Yes	Yes	No	Yes	Purulent	No	Natural	Necroscopy: specimens of intestinal contents taken after death										Cholera.
23	—	1	16	Yes	Yes	No	No	No	No		do										Do.
24	—	8	13	Yes		No	No	No	No		No	Moist	Moist	Bright blood and hyperemia							No cholera.
25	—	2	24	Yes	No	No	Yes	No	Yes	Diminished	No	Edema	Ascites	do	Normal	Congestion	Congestion	Normal	Edema		Do.
26	—	8	25	Yes			No	No	No		No	do	do	do	do	do	do	do	do		Do.
27	—	3	12	Slight	No		No	No	Yes		No	Moist	Moist	do	do	do	do	do	Normal		Do.
28	—	1	—	Slight	No	No	No	No	Yes	Natural	No	do	do	do	Congested	do	Congestion	do	do		Do.
29	—	2	—	Yes	No	No	Yes	No	Yes	Anuria	No	Edema	do	do	do	Albuminous degeneration	Albuminous degeneration	Inflamed	Congestion		Do.
30	—	9	—	Yes	No	Yes	Yes	Slight	Yes	Normal	Yes	Dry	Dry	Bright blood	do	do	do	Normal	Edema		Cholera.
31	—	2	17	Yes		No	Yes	Slight			No	Moist	Moist	Bright blood and hyperemia	do	Congestion	Congestion	Normal	Edema		Do.
32	—	1	8	Yes	Yes				Yes		No	Edema	Ascites	do	do	do	do	do	do		
33	—	1	—	Yes	Slight	No	Yes	Yes	Yes		No	Normal	Moist	Normal	Normal	Normal	Normal	Normal	Normal		
34	—	5	14	Yes	No	No	No	No	Yes	Scanty	No	Edema	do	Bright blood and hyperemia	Congested	Congestion	Congestion	do	do		
35	—	1	15		No	No	No	No	Yes	Anuria	No	do	Ascites	do	do	do	do	do	Edema		
36	—	2	19		No	No	No	No	Yes	Normal	No	do	do	do	do	do	do	do	Congestion, edema		
37	—	1	25	Yes	No	No	No	No	Yes	Scanty	No	Moist	do	do	do	Albuminous degeneration	do	do	do		
38	—	1	14	Yes	Yes	No	Yes	Yes	Yes		No	Edema	do	do	do	Congestion	do	do	Congestion		
39	—	3	5	Yes	No	No	No	No	Yes	Normal	No	Dry	Moist	Normal	Congested	Granulated	Congestion	do	Hemorrhages	Cerebral hemorrhage	
40	—	1	11	Yes	No	No	No	No	Yes		No	Edema	Ascites	Bright blood and hyperemia	Post-mortem decomposition					Ruptured	

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[1] Post-mortem decomposition.

TABLE VI.—*Cases of alleged meningitis.*

	Clinical data.									Pathologic findings.										
	Age.	Convulsions or convulsive symptoms.	Rigidity of neck.	Strabismus.	Fever.	Collapse.	Vomiting.	Rice water diarrhœa.	External appearance of cholera.[a]	Subcutaneous tissues.	Peritoneum.	Heart.	Lungs.	Spleen.	Kidneys.	Liver.	Intestines.	Meninges and brain.	Anatomic diagnosis.	Bacteriologic diagnosis.
	y. m. d.																			
1	3 — —	Yes	No	No	Slight	No	No	No	Yes	Dry	Dry	Normal	Congestion	Normal	Nephritis	Albuminous degeneration	Slight congestion	No inflammation	Cholera	Cholera
2	2 4 —	No	Yes	Yes	Yes	No	No	Feculent diarrhœa	Yes	do	do	Right, dilated	Congestion and œdema	do	Albuminous degeneration	do	Congestion	Congestion	do	D
3	— 10 —	Yes	No	Yes	Yes	No	Nausea	Yellow diarrhœa	Yes	do	do	do	do	do	Nephritis	do	Inflamed	Congestion, œdema	do	D
4	1 — —	Yes	No	Yes	Yes	No	No	No	Yes	do	do	do	do	do	do	Congestion	do	do	do	D
5	1 — —	Yes	No	Yes	Yes	No	No	Constipation	Yes	do	do	do	Congestion	do	do	do	do	do	do	Negr
6	3 — —	No	No	Yes	High	No	No	No	Yes	do	do	do	do	do	Congestion	Albuminous degeneration	do	do	do	Chole
7	3 — —	Yes	Yes	No	No	Cold extremities	Yes	Yes	Yes	do	do	do	do	do	Nephritis	Congestion	do	Congestion	do	B
8	— 6 —	No	No	Slight	Yes	No	No	No	Yes	do	do	do	do	do	Congestion	do	do	Congestion, œdema	do	Negr
9	2 — —	Yes	Yes	No	Yes	No	No	No	No	Moist	Moist	Normal	do	do	Nephritis	Normal	Normal	Suppurative congestion	Suppurative meningitis	D
0	— 10 —	No	No	No	Slight	No	No	Feculent diarrhœa	No	do	do	do	Normal	do	Normal	do	do	Normal	Undetermined[b]	D
1	— 5 —	Yes	Yes	No	Yes	No	No	Yellow diarrhœa	No	do	do	do	Pneumonia	Congestion	Nephritis	do	do	Congestion	Pseudo-lobar pneumonia[b]	No re
2	— 3 —	Yes	No	No	Yes	No	Yes	do	No	Dry	Dry	Soft	Congestion	Normal	Normal	do	do	Congestion, œdema	Undetermined	Negr
3	2 — —	Yes	No	Yes	Yes	No	No	No	Yes	do	do	Right, dilated	do	do	Nephritis	do	Inflamed	Congestion	Cholera	Chole
4	3 6 —	Yes	No	No	High	No	No	Yellow, liquid stools	Yes	do	do	do	Congestion and œdema	do	Congestion	Congestion	do	No inflammation	do	Negr
5	— 8 —	No	Slight	Yes	Slight	No	Nausea	No	Yes	do	do	do	Congestion	do	Albuminous degeneration	Albuminous degeneration	do	do	do	Chole
6	1 1 —	Yes	No	No	High	No	do	Greenish yellow stools	Yes	do	do	do	do	do	Congestion	do	do	Congestion	do	D
7	— 8 —	Yes	No	No	Slight	No	No	Greenish-yellow liquid stools	Yes	Post-mortem decomposition									Undetermined[c]	D
8	5 7 —	Slight	No	No	Yes	No	No	No	Yes	Dry	Dry	Right, dilated	Congestion	Normal	Albuminous degeneration	Albuminous degeneration	Inflamed	No inflammation	Cholera	D
9	— 4 —	Yes	Yes	Yes	Yes	No	No	No	Yes	do	do	do	do	do	do	do	do	Congestion	do	D
0	8 — —	Yes	No	No	Yes	No	No	No	Yes	do	do	do	do	do	do	do	do	No inflammation	do	D
1	1 — —	No	No	No	Slight	No	No	No	Yes	do	do	do	do	do	do	do	Congestion	Congestion	do	D
2	— 11 —	No	No	No	Yes	No	No	No	No	Slight œdema	Ascites	Right, dilated and hypertrophied	do	Congestion	do	do	Normal	Normal	Beriberi	Negr
3	5 — —	Yes	No	Yes	Yes	No	No	No	No	Moist	do	do	Congestion and œdema	do	Congestion	do	do	Congestion	do	D
4	— 8 —	Yes	No	Ptosis	Yes	No	Yes	No	No	do	Moist	do	Congestion	Normal	Albuminous degeneration	do	Slight congestion	do	do	D
5	— 2 —	Yes	Yes	Yes	Yes	No	No	No	No	do	do	do	Broncho-pneumonia	Congestion	do	do	do	do	do	D
6	— 4 —	Yes	Yes	Yes	High	No	No	No	No	Firm	Normal	Dilated and hypertrophied	Empyema	do	Congestion, albuminous degeneration	do	Normal	Suppurative meningitis	Meningitis	D
7	— 8 —	No	No	Yes	High	No	No	No	No	do	Moist	do	Congestion	do	Albuminous degeneration	do	do	Congestion	Beriberi	D
8	— 11 —	Yes	Yes	Yes	Yes	No	Nausea	No	Yes	Dry	Dry	Right, dilated	Congestion and œdema	Normal	Congestion	Congestion	Inflamed	Congestion, œdema	Cholera	No re
9	— 10 —	Yes	No	Yes	Yes	No	Yes	Soft, yellow stools	No	Moist	Ascites	Right, dilated and hypertrophied	do	Congestion	Albuminous degeneration	Albuminous degeneration	do	do	Beriberi	Negr
0	— 7 —	Yes	No	No	Yes	No	No	do	No	Firm	Normal	Normal	Congestion	do	Normal	Congestion	Normal	Slight congestion	Empyema	D
1	— 9 —	Yes	Yes	Yes	Yes	No	Yes	No	No	Moist	Ascites	Right, dilated and hypertrophied	do	do	Congestion	do	do	Congestion, œdema	Beriberi	D
2	2 7 —	Yes	Yes	Yes	Yes	No	Yes	No	Yes	Dry	Dry	Right, dilated	do	Normal	Albuminous degeneration	Albuminous degeneration	Inflamed	Congestion	Cholera	No re
3	— — —	No	No	No	Yes	No	No	No	No	Moist	Moist	Normal	Pneumonia	do	Congestion	Congestion	Congestion	do	Pneumonia	
4	— — —	Yes	No	Yes	Yes	No	Yes	Yellow	No	Firm	Ascites	Right, dilated and hypertrophied	Congestion œdema	Congestion	do	do	Normal	Congestion, œdema	Beriberi	
5	— — —	No	No	No	No	No	Nausea	No	No	Moist	Moist	do	Congestion	Normal	Congestion, albuminous degeneration	Congestion, albuminous degeneration	do	Normal	do	
6	— — —	Yes	No	No	No	No	No	No	No	do	Ascites	do	Congestion and œdema	Congestion	do	do	do	Congestion, œdema	do	
7	— — —	Yes	No	No	No	No	No	No	No	Dry	do	Normal	Congestion	Normal	Congestion	do	Inflamed	Normal	Acute colitis	

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[a] By [illegible] have reference to [illegible] presenting the following: A haggard drawn expression of the face with sunken, half open eyes. The finger nails are bluish in color, the skin of the fingers is shriveled and wrinkled, and the skin covering the body is drawn and tight, as a rule.
[b] Greatly em[illegible] a[illegible] due to nonassimilation of [illegible]
[c] Post-mortem [illegible]

TABLE VI.—*Cases of alleged meningitis.*

Clinical data.								Pathologic findings.									
Age.	Convulsions or convulsive symptoms.	Rigidity of neck.	Strabismus.	Fever.	Collapse.	Vomiting.	Rice water diarrhœa.	External appearance of cholera.[a]	Subcutaneous tissues.	Peritoneum.	Heart.	Lungs.	Spleen.	Kidneys.	Liver.	Intestines.	Meninges and brain.	Anatomic diagnosis.	Bacteriologic diagnosis
y. m. d.																			
3 — —	Yes	No	No	Slight	No	No	No	Yes	Dry	Dry	Normal	Congestion	Normal	Nephritis	Albuminous degeneration	Slight congestion	No inflammation	Cholera	Cholera
2 4 —	No	Yes	Yes	Yes	No	No	Fæculent diarrhœa	Yes	do	do	Right, dilated	Congestion and œdema	do	Albuminous degeneration	do	Congestion	Congestion	do	Do
— 10 —	Yes	No	Yes	Yes	No	Nausea	Yellow diarrhœa	Yes	do	do	do	do	do	Nephritis	do	Inflamed	Congestion, œdema	do	Do
1 — —	Yes	No	Yes	Yes	No	No	No	Yes	do	do	do	do	do	do	Congestion	do	do	do	Do
1 — —	Yes	No	Yes	Yes	No	No	Constipation	Yes	do	do	do	Congestion	do	do	do	do	do	do	Negati
3 — —	No	No	Yes	High	No	No	No	Yes	do	do	do	do	do	Congestion	Albuminous degeneration	do	do	do	Choler
3 — —	Yes	Yes	No	No	Cold extremities	Yes	Yes	Yes	do	do	do	do	do	Nephritis	Congestion	do	Congestion	do	De
— 6 —	No	No	Slight	Yes	No	No	No	Yes	do	do	do	do	do	Congestion	do	do	Congestion, œdema	do	Negati
3 — —	Yes	Yes	No	Yes	No	No	No	No	Moist	Moist	Normal	do	do	Nephritis	Normal	Normal	Suppurative congestion	Suppurative meningitis	Do
— 10 —	No	No	No	Slight	No	No	Fæculent diarrhœa	No	do	do	do	Normal	do	Normal	do	do	Normal	Undetermined[b]	Do
— 5 —	Yes	Yes	No	Yes	No	No	Yellow diarrhœa	No	do	do	do	Pneumonia	Congestion	Nephritis	do	do	Congestion	Pseudo-lobar pneumonia[b]	No rep
— 3 —	Yes	No	No	Yes	No	Yes	do	No	Dry	Dry	Soft	Congestion	Normal	Normal	do	do	Congestion, œdema	Undetermined	Negati
2 — —	Yes	No	Yes	Yes	No	No	No	Yes	do	do	Right, dilated	do	do	Nephritis	do	Inflamed	Congestion	Cholera	Choler
3 6 —	Yes	No	No	High	No	No	Yellow, liquid stools	Yes	do	do	do	Congestion and œdema	do	Congestion	do	Congestion	No inflammation	do	Negati
— 8 —	No	Slight	Yes	Slight	No	Nausea	No	Yes	do	do	do	Congestion	do	Albuminous degeneration	Albuminous degeneration	do	do	do	Choler
1 1 —	Yes	No	No	High	No	do	Greenish yellow stools	Yes	do	do	do	do	do	Congestion	do	do	Congestion	do	Do
— 8 —	Yes	No	No	Slight	No	No	Greenish-yellow liquid stools	Yes	Post-mortem decomposition									Undetermined[c]	Do
5 7 —	Slight	No	No	Yes	No	No	No	Yes	Dry	Dry	Right, dilated	Congestion	Normal	Albuminous degeneration	Albuminous degeneration	Inflamed	No inflammation	Cholera	Do
— 4 —	Yes	Yes	Yes	Yes	No	No	No	Yes	do	do	do	do	do	do	do	do	Congestion	do	Do
8 — —	Yes	No	No	Yes	No	No	No	Yes	do	do	do	do	do	do	do	do	No inflammation	do	Do
1 — —	No	No	No	Slight	No	No	No	Yes	do	do	do	do	do	do	do	Congestion	Congestion	do	Do
— 11 —	No	No	No	Yes	No	No	No	No	Slight œdema	Ascites	Right, dilated and hypertrophied	do	Congestion	do	do	Normal	Normal	Beriberi	Negati
5 — —	Yes	No	Yes	Yes	No	No	No	No	Moist	do	do	Congestion and œdema	do	Congestion	do	do	Congestion	do	Do
— 8 —	Yes	No	Ptosis	Yes	No	Yes	No	No	do	Moist	do	Congestion	Normal	Albuminous degeneration	do	Slight congestion	do	do	Do
— 2 —	Yes	Yes	Yes	Yes	No	No	No	No	do	do	do	Broncho-pneumonia	Congestion	do	do	do	do	do	Do
— 4 —	Yes	Yes	Yes	High	No	No	No	No	Firm	Normal	Dilated and hypertrophied	Empyema	do	Congestion, albuminous degeneration	do	Normal	Suppurative meningitis	Meningitis	Do
— 3 —	No	No	Yes	High	No	No	No	No	do	Moist	do	Congestion	do	Albuminous degeneration	do	do	Congestion	Beriberi	Do
— 11 —	Yes	Yes	Yes	Yes	No	Nausea	No	Yes	Dry	Dry	Right, dilated	Congestion and œdema	Normal	Congestion	Congestion	Inflamed	Congestion, œdema	Cholera	No rep
— 10 —	Yes	No	Yes	Yes	No	Yes	Soft, yellow stools	No	Moist	Ascites	Right, dilated and hypertrophied	do	Congestion	Albuminous degeneration	Albuminous degeneration	do	do	Beriberi	Negati
— 7 —	Yes	No	No	Yes	No	No	do	No	Firm	Normal	Normal	Congestion	do	Normal	Congestion	Normal	Slight congestion	Empyema	Do
— 9 —	Yes	Yes	Yes	Yes	No	Yes	No	No	Moist	Ascites	Right, dilated and hypertrophied	do	do	Congestion	do	do	Congestion, œdema	Beriberi	Do
2 s.	Yes	Yes	Yes	Yes	No	Yes	No	Yes	Dry	Dry	Right, dilated	do	Normal	Albuminous degeneration	Albuminous degeneration	Inflamed	Congestion	Cholera	No rep
) .			No	Yes	No	No	No	No	Moist	Moist	Normal	Pneumonia	do	Congestion	Congestion	Congestion	do	Pneumonia	
- s.				Yes	No	Yes	Yellow	No	Firm	Ascites	Right, dilated and hypertrophied	Congestion œdema	Congestion	do	do	Normal	Congestion, œdema	Beriberi	
No					No	Nausea	No	No	Moist	Moist	do	Congestion	Normal	Congestion, albuminous degeneration	Congestion, albuminous degeneration	do	Normal	do	
Yes						No	No	No	Ascites	do	Congestion and œdema	Congestion	Congestion	do	do	do	Congestion, œdema	do	
Yes						No	No	No	Dry	do	Normal	Congestion	Normal	Congestion	do	Inflamed	Normal	Acute colitis	

[a] By ... have reference to ... picture presenting the following: A haggard drawn expression of the face with sunken, half open eyes. The finger nails are bluish in color, the skin of the fingers is shriveled and wrinkled, and the skin covering the body is drawn and tight, as a rule.
[b] Greatly ... a.
[c] Post-mortem ... to nonassimilation ... too.

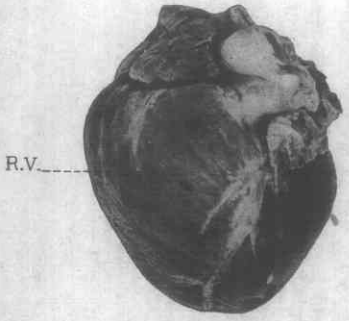

Fig. 1.

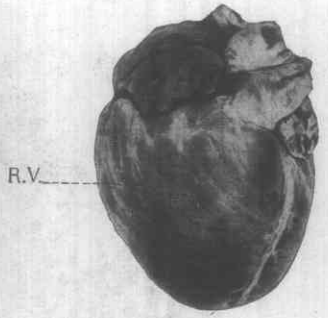

Fig. 2.

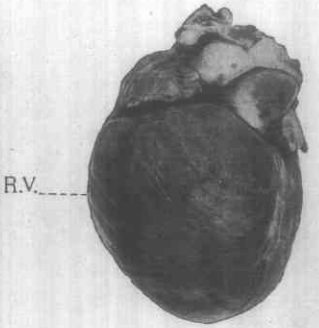

Fig. 3.

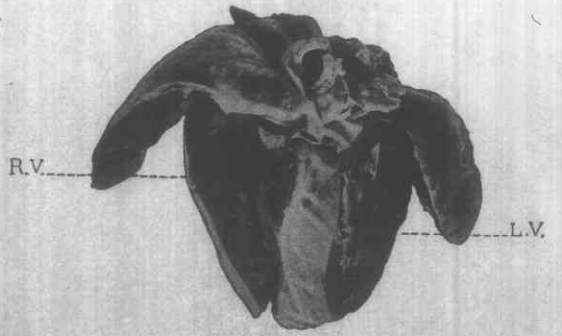

Fig. 4

PLATE I.

THE RELATIONSHIP OF FOOD TO PHYSICAL DEVELOPMENT.[1]

By D. McCay.[2]

"It is food that supplies the material for that perpetual series of transformations in which life consists, and it must be adequate in quantity and suitable in quality if these transformations, of so many different kinds, in so many different organs, are to proceed with that nicely balanced adjustment that is known as health." [3]

The question of the proper amount of daily food necessary to meet the physiological needs of the body is one that has occupied the attention of a great many workers since Chittenden stirred the nutritional pool. Believing that light would be thrown on the problem by a knowledge of the conditions that obtained in India, a series of investigations was undertaken to ascertain, if possible, the nutritive value of the different types of diet on which the teeming millions of India live. The inquiry soon resolved itself into one of determining the levels of nitrogenous metabolism attained on the different dietaries, and their effects on the physical development and well-being of the races investigated.

The dietaries being largely of a vegetable nature there is always an abundance of the carbohydrate element and a sufficiency of fat.

The first observations were made on students and others belonging to the Medical College, Calcutta, and also on some prisoners in the presidency jail. It was found that the average native of Lower Bengal on the ordinary diet of the province, namely, rice and *dal,* attains even a lower level of nitrogenous metabolism than Chittenden found to be quite compatible with health, bodily comfort, and the maintenance of strength and vigor. The observations made showed that students and members of the fairly well-to-do classes exist on a metabolism of less than 40 grams of protein per man daily. The great mass of the population is on an even lower scale than this. These results bore out Chittenden's views as regards the possibility of man existing on a protein content of the general diet less than one-third that of the ordinary

[1] Read at the First Biennial Meeting of the Far Eastern Association of Tropical Medicine held at Manila March 14, 1910.

[2] Captain I. M. S., Professor of Physiology, Medical College of Bengal, Calcutta.

[3] Sir J. Crichton-Browne.

standards, and, so far as they went, we freely admitted that the protein metabolism of the Bengali confirmed and corroborated his opinion.[4] It was when we tried to judge the effects of this dietary on the physical development of the race, the capacity of its individuals for manual labor, the condition of their blood and tissues, and, above all, their resisting power to disease and infection, that we were forced to part company from Chittenden and from the views he holds regarding the beneficial effects of a reduction of protein in the daily diet of mankind.

We showed the miserable standard of the Bengali's physical development, seemingly to be attributed to the low scale of protein absorption possible from their diet, by observations on students, prisoners, servants, and by an analysis of the records of the physical development of Bengali and Anglo-Indian students in the same college, under the same climatic conditions, doing the same work, but on a different diet. Without entering into any details of the work carried out on these lines, we may state that from the evidence brought forward, while admitting that it was quite possible for an individual or the members of a whole race to live on a metabolism of 6 grams of nitrogen daily, the results of this small intake on their general well-being, health, physical development, resistance to infection, and immunity from kidney disease[5] were not such as to confirm a belief in the sufficiency of Chittenden's standards. According to his views the metabolism of 0.12 gram of nitrogen per kilo of body weight is all that is necessary for the protein requirements of the body, which is practically the figure we obtained for the Bengali.

What are the effects on the physical development and general well-being of the people having this low level of nitrogenous interchange? The diet in Lower Bengal consists practically of rice and *dal* or pulse. It is an exceedingly bulky food when cooked, and, in order to provide for even the lowest limits of protein metabolism, a very large quantity has to be consumed; so large, as we have found, that the actual bulk interferes with its absorption.

The work on the Bengali showed, with regard to the chemical analysis of the blood, a higher percentage of water and a lower percentage of total solids and protein; the hæmoglobin was markedly reduced (about 75 per cent) and the blood pressure was on a distinctly lower level than that found among the Anglo-Indian students. We found that the results of these conditions were markedly to modify the physiological requirements of nutrition, and to a considerable extent to affect the growth and power of muscular contraction of the average individual, whose nitrogenous tissues are not given the option of drawing their nutritive material from so rich a source as more favored individuals do, nor have they the same opportunity of obtaining as free a supply of oxygen. We concluded from

[4] *Sci. Mem. Off. Med. & San. Dept.* Calcutta (1908), No. 34, 7, 8, 25, 28, 52.
[5] A point on which Chittenden lays great stress.

the study that the people on a diet from which only 37.5 grams of protein are absorbed live in a more or less chronic state of nitrogen starvation, leading to loss of body fat and tissue protein with an accompanying loss of vigor and strength and a comparatively low capacity for sustained muscular effort. From the evidence as to the physical development we came to the conclusion that the general physique of the Bengali is on a par with his diet, and that a close relationship exists between the poor physical development of this people and the meager protein absorption possible from the diet on which they subsist.

This was particularly well brought out by an analysis of the recorded weights, chest measurements, and heights of Bengali and Anglo-Indian students during the several years of attendance at one of the residential colleges in Calcutta. Under the same conditions but on different diets we found:

1. There was an increase of 7 kilos (14 pounds) in the average weight of Anglo-Indian and Eurasian students as compared with an average increase in body weight of 1 kilo (2 pounds) in the case of Bengali students on their diet, the observations being made over similar periods.

2. 42.8 per cent of the Bengali students showed a diminution in weight as compared with 2 per cent among the Anglo-Indian students. Among the former only 15.3 per cent gained weight continuously during the four years in residence, whereas practically all gained weight continuously among Anglo-Indian students.

3. The chest measurements bear out the same conclusions; the Bengali remains practically unaltered, whereas the Anglo-Indian increases his chest girth very considerably.

The diets on which these results were obtained are as follows:

Diet of Bengali students, in grams.		*Diet of Anglo-Indian students, in grams.*	
Protein (of which 9.3 were derived from an animal source)	67.11	Protein (of which 38.32 were derived from an animal source)	94.97
Carbohydrate	548.73	Carbohydrate	467.00
Fat	71.55	Fat	56.20

Comment on these results is unnecessary. They show very conclusively what may be expected in growing lads from diets respectively deficient and rich in absorbable protein.

Further evidence with regard to the physical endurance, capabilities of performing work, and the experiences of life insurance companies all place the Bengali on a low plane of physical development. The general consensus of medical opinion further shows that this power of resistance to disease is markedly inferior to that of the more highly fed European. Even in considering the incidence of renal disease, the facts do not bear out Chittenden's contention of the great advantage to the excretory organs of a low protein intake. Renal disease is much more common

among the ordinary working population of Bengal and, in combination with diabetes, very much more so among the higher classes than among Europeans in Europe or India. This is all the more remarkable in a country where scarlet fever is unknown and where the consumption of alcohol by the people is almost negligible.

The general conclusion to be drawn from the investigations on the metabolism of the Bengali is that his physical development[6] is only such as could be expected from the miserable level of nitrogenous interchanges to which he attains.

From the results of work carried out on the Behari and other tribes of the plains of Bengal and United Provinces we obtained undoubted evidence of superiority in physique and muscular development and, what is also very noteworthy, a distinctly greater degree of vivacity, briskness, and sprightliness of manner. The body weight is also on a higher scale, being on the average 5 to 7.5 kilos greater than is the case with the Bengali. The ordinary working population of Bengal is characterized by a want of vigor, a slackness, tonelessness, general slowness of reaction, and other physiological attributes of torpor difficult to describe, detect, and measure. Self-absorption and want of interest in the incidents of everyday life, little power of attention, observation, or concentration of thought are some of the attributes of all but the better classes and of the better fed among the Bengalis. The Behari and the inhabitants of United Provinces do not show these characteristics to anything like the same extent. What kind of dietary do these people live on? The Behari lives on a mixed diet of wheat, maize, rice, and *dal;* the inhabitants of Agra and Oude live largely on wheat, different millets, barley, maize, and *dal*. Without going into details of the different foodstuffs it may be accepted that the ordinary workingman has a diet from which he can absorb at least 9 grams of nitrogen per day. Rice as a rule forms no part of the dietary. This would give a metabolism for the different races included above of from 0.15 to 0.18 gram of nitrogen per man daily, a quantity that Chittenden would consider excessive, and it is fully 20 to 50 per cent superior in its most important element, nitrogen, to the dietaries of Lower Bengal. As we have already stated, the physical fitness and development of these races are much superior to the same characters obtaining in Bengal, and, as far as the evidence goes, the latter would appear to obey the biologic law, namely, their protoplasmic development is a function of the absorbable protein of the diet.

Now the question arises, Are there any other factors except differences in diet that will satisfactorily account for this higher standard of physical development and general well-being?

[6] The actual amount of protoplasmic tissues as distinguished from fatty tissue.

We believe the presence of wheat, maize, millets, etc., replacing the bulky rice, which is of low nutritive value, sufficiently explains the situation; however, we shall examine some of the objections which have been brought forward against the view that defective nutrition is the result of a low protein intake.

Doctor Kellogg, a strong advocate of vegetarianism, criticised our finding regarding the important rôle played by diet, and especially by protein, in the nutrition of the Bengali, as follows:

> The weakest part of the report from my standpoint is the remarks which the investigator makes in relation to the defective nutrition resulting from the low protein dietary. I do not think it is at all fair to attribute the lack of endurance often seen among Indians to the low protein diet. There are so many factors which certainly should be taken into consideration. Among these are their sexual excesses, the depressing effects of the very hot, damp climate in which they live, and which predispose to lack of exercise, the injurious effects of excessive, prolonged exposure to the actinic rays of the sun. Still another factor of importance is the immature age at which these people usually marry. Many of the Indians, however, are strong and robust people. I understand that an Indian regiment made up entirely of natives is the finest lot of men in His Majesty's service.

We agree that these causes have undoubtedly an influence in retarding growth and lowering the general standard of physique, and, if there were no means of estimating their effects, it would be very difficult to say that they are not quite sufficient, as Doctor Kellogg believes, to account for the relative difference between Europeans and the poorer developed natives of India.

Many objections to Doctor Kellogg's views immediately arise, for instance, as regards climate and the actinic rays of the sun. Europeans, Eurasians, and the better-fed Bengalis are all equally exposed to these influences, yet retain their energy. We have made inquiries regarding sexual excess, and, while masturbation probably is more prevalent among the Bengalis, excessive sexual congress is chiefly practiced by the better classes, who have the means and energy to satisfy their desires. Immature marriage is undoubtedly a factor, but there are customs which have a tendency to neutralize its ill effects: The husband and wife do not live together until the wife reaches puberty—the husband is usually several years older than the wife, and the latter spends about one-third of each year with relatives away from the husband. Another effort of nature to maintain the standard is the relatively higher death rate amongst the children born early in marriage. However, while these replies to Doctor Kellogg's criticisms are important in showing that his objections do not cover the whole ground, it is evident they are too indefinite to be measured and appraised at their full value. They would, even at the best, be only a matter of opinion, one school of thought looking on them from a point of view different from that of another.

In order to obtain definite and precise knowledge as to the effects of diet *per se*, we have extended our inquiries to different tribes and races in which the several factors enumerated by Doctor Kellogg are common to all, the dietaries forming the main point of difference. In this way we can eliminate the influence of the sun's rays, early marriages, climate, sexual excess, etc., in fact everything except the rôle played by diet, or, more particularly, absorbable protein, in the conditions that go to make one class superior to another, or one tribe or race superior to another tribe or race.

We find the different tribes and races whose characteristics we have been discussing inhabiting the plains extending from the sea opposite Bengal to the base of the mountains bordering Bengal, the United Provinces and the Punjab on the north. Now, all the factors Doctor Kellogg laid stress on are present amongst these people. The climate from Behar to the mountins, north and northwest, is to all intents and purposes the same; early marriages, sexual excesses, actinic rays of the sun, etc., are all even more in evidence than in Bengal; yet when we come to investigate the different attributes that go to make up a man, we find that there is an ascending scale of physique and manly qualities among the inhabitants extending from Lower Bengal to Behar and from Behar to the Provinces of Agar and Oude. There is an ascending scale of body weight and particularly of the protoplasmic tissues. A decided change in the demeanor and general appearance takes place as we pass from plain to plain, the people becoming brighter, fitter, and more energetic in their movements.

It may therefore be concluded that diet appears to play the principal part in the formation of the respective characteristics and general bearing of these races. The difference in diet is the substitution of an assimilable form of protein in the shape of nonbulky foodstuffs for a bulky material (rice) of low nutrition value, or, translated into its ultimate effects, the metabolism of 9 grams of nitrogen instead of 6 grams as found in Bengal.

With regard to the last part of Doctor Kellogg's criticism we may say a few words.[1]

It could not be expected that a person who had never been in India would be in a position to differentiate between the races; so that the inclusion of the Bengali among the great fighting races is, therefore, quite easily understood; nor would the point call for any comment were it not that true facts afford still further evidence of the important rôle played by diet. The Bengali has never, in modern times, so far as we are aware, been recruited for the fighting line, and although many regiments are, or were, called Bengal infantry, Bengal cavalry, etc., not a single man carrying a rifle could claim Lower Bengal as his place of

[1] "I understand that an Indian regiment made up entirely of natives is the finest lot of people in His Majesty's service."

birth. We have no desire to elaborate the point, but the question arises, Why is the Bengali unfit for the fighting line when other inhabitants of the plains exposed to, and suffering from, all the disabilities that Doctor Kellogg enumerates, but living on a superior diet, are capable of exhibiting the firmest courage and of maintaining untarnished the great fighting traditions of their race? Thus we have the Sikhs, famous throughout the world for their endurance and fighting qualities, inhabitants of the hottest plains of India, yet men of splendid physique and full of energy; the Dogras, Jats, Rajputs, all well known for their own special qualities on the Indian frontier, or wherever courage, endurance and determination are called for. These races labor under the disadvantages advanced by Doctor Kellogg to explain the relatively poor development and lack of endurance of the Bengali, but we have obtained no evidence from a study of these people of the correctness of his opinions.

Even in the various classes of these and allied races differences in physique, muscular development, hardiness and all those qualities that go to make up the perfect soldier can be detected. We believe that diet, and particularly the level of nitrogenous metabolism attained, has an immense influence on the formation of those most desirable characteristics of the races whence is drawn our best fighting material.

We have made extensive inquiries on the same lines among the hill tribes of Bengal and have no hesitation in asserting that the evidence obtained confirms and corroborates the view put forward as to the rôle of assimilable protein and its determining influence on the physical development and character formation of a people. We took up the different tribes inhabiting the hills around Darjeeling and contrasted, as far as possible, the physique and general characteristics of the several races. While there is no doubt but that climate has a great deal to do with the higher scale of general development and capabilities of these tribes as compared with those of the plains, this is not the whole story as is brought out by a comparison of the several classes living under practically identical conditions, but with a difference in diet forming the one conspicuous influence on their respective attributes.

We do not wish at present to go into details of the work carried out on these hill tribes. Suffice it to say that the Bhutia, by far the most capable of these people in those occupations requiring great muscular exertion, attain a nitrogenous metabolism much higher than that of any other tribe, or, indeed, any other race we have investigated. Just as was the case with the inhabitants of the plains, so we find with the races in the hills that variations in the level of nitrogenous metabolism appear to be the determining factor of the several causes that go to relegate, fix, and maintain the position of a tribe or race in the scale of mankind.

The close relationship between the nutritive value of the several

dietaries and its influence on the physical development of the different tribes and races that we have investigated is clearly brought out in the following scale of the degree of nitrogenous interchanges:

	Grams of nitrogen per kilo body weight.
First. Bhutias	
Nepalese Bhutias	[s] 0.42
Tibetan and Bhotan	[s] 0.35
Sikkim Bhutias	0.25
Second. Nepalese	0.18–0.25
Third. Behari	0.15
Fourth. Bengali and Ooriya	0.116

We have only taken into account the inhabitants of Bengal, as the work in other provinces is not yet completed; but nothing we have learned in further investigations has tended to contravert the opinion we have expressed; on the contrary, the more the subject has been gone into the stronger the evidence becomes of the correctness of our views. Every possible cause, *except diet,* has been put forward as offering a complete explanation of the inferior capabilities of the Bengali as compared with those of the great races of the plains of India. We have discussed these hazy, ill-defined influences, and, while admitting the probable force of some of them, have eliminated them by contrasting races in which all the factors are identical, but in which diet alone forms the distinguishing element, or, more correctly, in which the level of protein metabolism forms the great line of demarcation. We conclude from the studies that absorbable protein is the all-important element in the physical development and general well-being of mankind.

[s] Diet very highly animal.

UNSOLVED HEALTH PROBLEMS PECULIAR TO THE PHILIPPINES.[1]

By VICTOR G. HEISER.[2]

Many of the modern problems of hygiene and sanitation are the same the world over, whether found in tropical or temperate zones. There are certain characteristic phases of tropical hygiene and sanitation, however, which have received most gratifying attention in recent medical literature and which we can not but interpret as showing a very general interest and desire to make these portions of the world compare in healthfulness with those heretofore believed to be more favorably situated. However, in addition to these general problems, common to all tropical countries, there are additional difficulties and handicaps peculiar to each country or people, which have required special consideration.

It is the object of this paper not only to present the problems and theories with which you are already familiar, for it is desirable that you should know what we also are doing along these lines, but more especially to put before you the peculiar conditions which have seemed to hinder our more rapid progress and which are still blocking the way to better sanitation and hygiene in the Philippine Islands.

In general, we have first a poverty-stricken people with a poor physical inheritance, a people strongly imbued with superstitions and habits the antithesis of the simplest health doctrines and practices, a people lacking ambition productively to till the fertile soil, a people the masses of whom are apparently content in their ignorance and poverty and resigned to and uncomplaining of their many ailments. Work among them is handicapped by the inaccessibility of many of the islands and the nature of the roads, which, although being improved at a remarkable rate, are yet unfit for travel in many instances during portions of the year. It is further handicapped by the lack of a common language, for as many as fifty or more dialects are spoken among the *tao* or peasant

[1] Read at the first meeting of the Far Eastern Association of Tropical Medicine, held at Baguio, P. I., March 14, 1910.

[2] Passed assistant surgeon, United States Public Health and Marine-Hospital Service; Director of Health for the Philippine Islands; and Professor of Hygiene, Philippine Medical School.

classes, to whom neither Spanish nor English is intelligible. It is handicapped by the lack of a sufficient number of medical employees who are sufficiently interested in the cause of humanity to undergo the innumerable hardships and discomforts which accompany most of the medical service in the provinces. Untold credit is due those who are at present carrying on the work, the burdens of which fall more heavily upon them because of their limited numbers. We are also handicapped by a treasury of which it is quite possible at times to see the bottom, so that it is not put to us, "do all you can regardless of expenditure," but "your expenditures must not go beyond this or this; do the best you can with the funds available." So our very largest problem is that of discrimination: Shall we devote our energies to this or that question; we cannot do all; shall we attempt this one and abandon that, or do a little toward each? Thus far it has been our policy to do a little toward each, although the problems are many and great.

Malaria.—For instance, the malaria problem alone is a very large one. In the Province of Ambos Camarines, which has a population of 233,472 persons, there were 745 deaths from this disease reported for the year ended June 30, 1909. A conservative estimate of 10 cases for each death with an illness of ten days makes 74,500 days lost, which, valued at only 50 centavos Philippine currency or 25 cents United States currency a day, would amount to 37,250 pesos or 18,625 dollars United States currency a year. This being for only 1 out of 30 provinces, we can conservatively multiply this sum by 20, which would make an economic loss to the country of 745,000 pesos. If a human life can be economically valued at 1,000 pesos or $500 or £100 in this country, making deductions for children, etc., we find ourselves confronting a loss of at least 5,000,000 pesos per annum from this disease alone, and yet this represents but a small portion of the total loss which can be attributed to preventable diseases.

The number of malarial cases has been greatly decreased, however, by instituting drainage wherever possible, by the free distribution of quinine, and by campaigns of education, particularly in the schools, where pupils are taught the value of mosquito nets, the danger from mosquitoes, and how they may be destroyed, etc.

Mosquitoes.—There recently has been an active newspaper agitation concerning the eradication of mosquitoes in Manila, but few persons realize the obstacles that block our way in such an undertaking. Manila is on low land, much of its area being in fact below sea level. To bring the city to a drainable level would be a necessity in such a proposition, and is estimated to cost 4,000,000 pesos at least. When we reflect that the annual income of the city is under 3,000,000 pesos we naturally pause before recommending a measure so financially disproportionate, especially when some authorities consider such an undertaking problematical and more especially since there are practically no reliable data on

hand to show that the mosquitoes of Manila are more serious than a physical annoyance. We can well imagine how a similar proposition would be received in the United States or any other country. Should we expect it of these Islands, overburdened as they are with more acute problems? It seems to the writer more logical to begin mosquito elimination in Manila, at least, by educating the individual householder to make the breeding of mosquitoes on his premises impossible, and then by an organized inspection service to enforce regulations which will compel the use of such knowledge. In the meantime, a definite engineering project should be adopted and carried out in a limited way. Drainage and filling and oiling might be tried with the regular sanitary corps as far as possible.

Water supply.—The water supply of the Philippine Islands is another serious question. All surface waters found in the Islands except the thermal waters, or those strongly charged with certain minerals, are infected with amœbæ. Improved health conditions in Manila can to a certain extent be traced to the new city water supply which now comes from a comparatively uninhabited watershed, but even as tap water it should be boiled for all but the most ordinary purposes. Fortunately, artesian-well water is as a rule free from amœbæ, and is in every way an ideal drinking water. In towns where artesian well-water is almost exclusively used the death rate has fallen 50 per cent. These wells are being drilled as rapidly as possible, but there are many localities where they are impracticable, so that the question of how to make available for the people an unlimited and safe water supply, exclusive of artesian-well water, must be considered one of our unsolved problems.

Disposal of excreta.—We are still pondering the practical disposal of excreta. Many plans have been proposed, all more theoretical than practical. Of those who are familiar with local conditions and who realize the resources and limitations of the average community, but who are seriously searching for a practical solution, none as yet has put forth a well-defined scheme.

In large communities where water carriage of sewage is possible septic tanks are used successfully. In communities in which cholera has prevailed the pail system, the digging of pits and covering of excreta with lime or clean earth at regular intervals, has been found effective, but the cost of maintenance and inspection as a regular measure is prohibitive and only warranted by emergency conditions.

Some years ago the writer suggested a plan of installing a pail system with an after treatment of the night soil which would render it suitable for fertilizing mulberry trees, thus promoting the silk industry, the income from which would in a short time place this particular sanitary measure upon a self-paying basis.

The plan followed in many oriental countries of letting out private contracts for the collection of night soil from private residences is not

believed to be a safe one. It is an established custom to use such night soil for fertilizing vegetables, and it is believed that the consumption of raw vegetables thus fertilized has had much to do with the spread of amœbic dysentery, cholera, hookworm, and other intestinal diseases.

Smallpox.—Smallpox, once so formidable a proposition to us, has at least been reduced to insignificant proportions. Over 6,000,000 persons have been vaccinated by the Bureau of Health within the past five years. The unvaccinated are in remote regions where as yet it has been found impossible to convey vaccine in a potent condition.

The ordinary glycerinized lymph at present in use will not keep for more than seven to ten days at the temperature which prevails here. As many of the sections to be reached are in traveling time from two to three weeks away from the point to which ice can be sent, or where cold storage is available, it is obvious that a vaccine is necessary which will retain its potency for a longer period of time than any now obtainable. Vaccine in powdered form has been tried, also dry points, but the percentage of success is so small and danger of infection so great that their use is restricted.

The acute phase of this problem then is either to manufacture a more effective vaccine or to find a way of transporting it successfully. Observations point to the conclusion that ordinary cowpox vaccine is not as effective among the dark as among the white skinned races. The writer has personally observed that out of more than 100 cases of smallpox or varioloid among white people not one case occurred in a person who had been vaccinated within five preceding years, while there have been many cases of smallpox among Filipinos of whose successful vaccinations within one preceding year there could be little doubt.

It has been interesting to observe a demonstration of this at Bilibid Prison, where all prisoners are vaccinated upon admittance, and regularly once a year or oftener thereafter. Yet smallpox has made its appearance there each year, and many cases have occurred in persons who show the typical pits accepted as characteristic of previous attacks of smallpox.

Tuberculosis.—Tuberculosis is another of our problems. We estimate that it claims as many victims as in other portions of the globe, and it will require the same activity here as elsewhere to hold it in check. The introduction of out-patient tuberculosis dispensaries, the construction of shacks in the mountains, the opening of night camps near Manila, arrangement for the hospitalization of the helpless sick and prophylactic instruction in the public school, the usual methods in fact that are employed elsewhere, are now under way here; but the tuberculosis problem has its peculiar and complicating features in the Philippines, namely, the unsuitable dietary of the people, their peculiar superstitions concerning the contraction of the disease, their almost unshakable fear of night air as a poisonous thing, a fear which has kept their houses tightly closed at

night for generations past, their habit of chewing betel nut which has made the custom of expectorating in public and private a universal and we sometimes fear an incurable habit. Added to this is their utter resignation to the disease as a thing incurable and inevitable. Therefore, not only have we the ordinary preventive and curative measures against tuberculosis to organize and enforce, but we must devise ways of cooking and preparing native products into a suitably nourishing and popular dietary, and then educate the masses not only to the ways of adopting these reforms, but also to an earnest desire for them. They will have to be first cured of their superstitions, which is as great a task as converting them to a new religion; houses will have to be opened at night, betel nut chewing gradually abolished, and then a gigantic antispitting crusade begun, and, last of all, comes the Herculean task of rousing them out of their inertia and convincing them that not only is tuberculosis curable, but that they are responsible for the spread of the disease and able to themselves accomplish the cure.

Cholera.—Cholera is still with us in spite of the active measures which are constantly being taken to eradicate it. Although we have so far been successful in promptly suppressing outbreaks whenever they appear, yet it is constantly occurring in sporadic form over widely separated sections of the Islands. The puzzling question is this: When no possible connection with any previous case is discoverable, where are the cases of cholera contracted that appear so spasmodically over these widely separated regions? Whether it is lying dormant during the periods of time when no cases are reported is yet to be determined. Some years ago the writer drew attention to the fact that logical deduction indicated that there was a morphological change in the cholera organism which made it difficult to recognize at certain stages. Research work done in the meantime strengthens this view. There is also a strong probability that cholera carriers are responsible for some outbreaks; yet the fact remains that the disease appears frequently at places in which its origin can not satisfactorily be proved.

Plague.—Plague at present is a stranger to the Philippines. Measures for its eradication were begun in 1900, but it was not until the heretofore-described scheme of dealing with rats was used that the disease disappeared. Since April, 1906, no cases have been found in human beings, and no cases in rats since 1907.

However, on account of the close proximity of China, where plague seems to appear every year, special precautions are constantly taken at our ports in order to prevent the reintroduction of this disease. The question of course is how safely to accomplish this with a minimum amount of annoyance and loss to the shipping interests and to the traveling public. The present plan is to fumigate all vessels from infected ports twice annually and to keep the interisland vessels free

from rats and vermin by systematic fumigation, in order that the plague may at once be checked if by any accident introduced. Wharves have been made rat proof, and vessels where docked are required to use rat funnels in order to keep rodents from gaining access to the shore. At the port of departure for these Islands vessels are inspected by medical officers of the United States in order to ascertain that there is no plague aboard.

Typhoid fever.—While cases of typhoid fever are undoubtedly contracted in the Philippines, yet the disease can hardly be said to be prevalent here. To prevent its establishing a foothold, regulations were prepared for the disinfection of excreta from such cases, for the protection of water supplies, and regulations concerning inspection and sale of milk; also sanitary measures for the eradication of flies were undertaken. Whether further and more stringent precautions could be taken at this time, or are practicable, is a question for consideration.

Infant mortality.—The subject of infant mortality is a vast one. In Manila approximately one-half the total number of deaths occurs in children under one year of age. From papers read at this meeting and previously it is obvious that the largest share of it is due to improper nourishment. The poverty of the people makes properly marketed cow's milk, either fresh or canned, an impossibility unless given in the form of charity. This for the great mass of people is not only impossible but undesirable. How to bring a cheap supply within the reach of the poorer classes seems to be the acute phase of the infant-mortality problem. The raising of goats would seem to be the solution. Already experiments in breeding a hardy variety of milk goats have been inaugurated. This problem must not be abandoned, but be rapidly pushed to a solution, for if left unsolved it involves the heaviest mortality we are at present facing.

Putrefactive changes in foods.—Another complicating feature and cause of illness in the Tropics, particularly in the Philippines, is the putrefactive changes in nitrogenous foods which take place so rapidly in warm climates. The problem is either to provide ways properly to preserve such foods or to find suitable substitutes which will enable us to eliminate them from our tropical dietary.

Insanitary habits.—The food question brings in its trail another problem that is peculiarly ours and which we know to be the largest factor in the transmission of cholera and intestinal diseases. This is the habit of eating with the fingers. Proper example has done much and the distribution of literature on the subject has helped; but the masses are as yet untouched by either example or precpt, and we see years of discouraging struggle ahead of us before they can be broken of so fixed a habit, the menace of which is as yet entirely beyond their comprehension.

Poor statistical information.—So much for the particular problems.

The entire situation is hindered by our inability to secure proper statistical information. This is due to a lack of officials in the provinces sufficiently skilled to make reliable reports on the causes of death. Whether the municipal officials can be trained and educated to do this remains to be seen. As stated in the beginning, our work is first one of discrimination, a placing of our heaviest artillery where the enemy is strongest. This we can not always determine on account of the inaccuracy and incompleteness of available data.

SUMMARY.

To summarize, it is to be understood that the health of these people is the vital question of the Islands. To transform them from the weak and feeble race we have found them into the strong, healthy, and enduring people that they yet may become is to lay the foundations for the successful future of the country. But it is not alone the problem of the Bureau of Health; it is an economic and educational question as well. Every branch of the Government has its part to perform, and coöperation is essential. Good roads; agricultural improvements; the elimination of rinderpest and other animal diseases; the general development of the country, which will gradually bring about a better standard of living; education, particularly along the lines of hygiene and sanitation (to which we give all the aid possible, but for the dissemination of which we will have to depend upon the teachers and the public schools); the special training of the young men and women of the Islands in the professions of medicine and nursing—all the foregoing factors, with which we, as a Bureau, have nothing to do, are as important to the health conditions of the Islands as is the actual holding in check of epidemics and disease, the sanitary inspections, enforcement of regulations, etc., the opening and maintenance of hospitals throughout the Islands, and the various other things for which the Bureau of Health is directly responsible.

The Government is not a rich one. How to do the most and the best with a limited income is still an acute question. You can see what an enormous proportion of that limited income it would take to carry out successfully any one of the various health projects enumerated. To give thorough attention to a particular one would involve an unwarranted neglect of the rest. Hence we have concluded we must do the best we can with the entire proposition, going slowly but making headway each year, each month, perhaps each day.

It should be remembered that much of our appropriation is consumed in ways unusual for a health bureau. The maintenance and management of general and insane hospitals, orphan asylums, homes for the aged, etc., falls to our lot and is no small burden. We are practically cleaning up these Islands, left foul and insanitary and diseased by

generations of hygienically ignorant peoples. We are stamping out the conflagration of disease started long before American occupation, and not until it is stamped out can we look forward to the modern problems which come so temptingly before us. And so, much of our time, money, and effort is being constantly consumed in works, the glory of which is still behind the clouds. We are draining the land, as it were, before beginning the constructive health projects which are going to make these people the strong and healthy race we intend them to be.

THE PARTHENOGENESIS OF THE FEMALE CRESCENT BODY.

By H. M. Neeb.[2]

As is well known, the parthenogenesis of the tertian gamete was first observed by Schaudinn in a patient, Mrs. Kossel, and was accurately described by him. This discovery is very important as it gives a clear and natural explanation of the cause of relapses in malaria, particularly in persons who have long since left the Tropics and are no longer exposed to active infection. This observation of Schaudinn was afterwards confirmed by Doctor Von Hilst Karrewly and then by Doctors Merz and Blüml.

It seemed highly probable that a metamorphosis of the tropical and quartan parasites would occur in a similar manner and should be sought for in a similar way. When I was in charge of the civil medical service at Koeta-Radja, during the hours of free consultation, I often had the opportunity of preparing blood-smears from natives who had contracted fever and had never been treated with quinine. Many of these patients were suffering from a severe infection with tropical malarial fever. For a long time my investigations were fruitless. However, two and a half years ago I examined blood-smears from a Bengalese and discovered the particular forms of parasites shown in Plate I, figs. 1, 2, and 3.

After an exhaustive consideration of every conceivable hypothesis, I finally came to the conclusion that I probably had encountered parthenogenesis in the female tropical gamete. However, I hesitated to publish such a discovery without accurate verification and confirmation by competent authorities on malaria; therefore, I took the opportunity, during my furlough, to have my slides examined in Europe at Bordeaux and Hamburg.

In the former city, Professor Le Dantec declared the forms represented by figs. 1 and 2 to be sporulation parasites, but quite different from those which are found in the common schizogenesis of the tropical parasites. However, he went no further than to state that in every case they were derived from a gamete.

[1] Read at the first meeting of the Far Eastern Association of Tropical Medicine, held at Manila, March 10, 1910.

[2] Medical officer of the first class, delegate from Her Majesty's Government of the Netherlands Indies.

The experienced protozoölogist, Doctor Von Prowazek, of the "Institut für Schiffs and Tropenhygiene" at Hamburg, declared the forms shown in figs. 1 and 2 to be parthenogenetic ones of the female crescent bodies. Professor Nocht found figs. 1 and 2 to be very interesting. He had never seen such forms, and, after due consideration, agreed with me that we are dealing with a case of parthenogenesis. Both the temporary assistant, Doctor Rodenwaldt, of the army, and Doctor Gonder, assistant to Doctor Von Prowazek, suggested it to be barely possible that the forms represented two microgametocytes, in which the chromatic mass had just divided to produce the microgametes, which would have been expelled forthwith. I believe I can bring forward a number of well-founded objections to this view, as will be seen from the discussion below. Doctor Werner was of the opinion that an exact critique was scarcely possible, because the blood-smears had been stained with Giemsa solution to which a small quantity of a solution of potassium carbonate had been added, and under such circumstances it was not inconceivable that other portions of the cell and of the parasite might also have been stained in the same manner as the chromatic substances. Doctor Mayer also found the forms under discussion to be very peculiar, but hesitated to express a definite opinion regarding them.

The interpretation of the parasitic forms which I demonstrated was still difficult, even after such authorities on malaria as Le Dantec, Nocht, Von Prowazek and others had examined the slides. I therefore determined, to the best of my ability, further to elucidate this point by preparing colored plates of the segmenting parasites, accompanied by a detailed description of the latter. The facts regarding the preparation of the specimens are as follows:

To the best of my recollection the Bengalese patient already mentioned came to me during my consultation hour. He appeared to be very feeble, was anæmic and cachectic. His temperature was between 38° and 39°C. The liver and spleen were enlarged. He had never taken quinine.

I prepared two slides in the usual way from a drop of blood. The blood on the slides was congealed in a few seconds by rapidly swinging them; then the preparations were immediately fixed with methyl-alcohol and afterwards stained for two hours with Giemsa solution (Grubler, Leipzig), 1 to 20, mixed with two or three drops of potassium carbonate, 1 to 1,000.

Beautiful examples of Maurer's *"perniciosa granules"* could immediately be observed on the infected red blood corpuscles. The corpuscles themselves were not enlarged and they had the usual red color. One or occasionally two tropical rings were generally to be found in the infected chromocytes and these were as a rule grown. However, the majority showed the peculiarity of containing one very large and, in certain instances, a double chromatic mass, the protoplasmic ring being proportionately very broad and coarse. I believe that many of these forms should be considered as young gametes, which later would have become crescent bodies.

Moreover, many crescent bodies could be found, for the most part female, which were almost full grown, or had recently attained maturity and which were still inclosed, more or less, in membranes of different form and of a dark, brick color. The envelope was very distinctly

recognizable as the stroma of the chromocyte. In addition, a slight polychromatophilia was present; basophilia could not be observed. However, there were peculiar, very large, retiform, brick-colored cells (macrophages from the spleen) and also a few macrocytes.

After careful and repeated examination of the blood preparations, I arrived at the conclusion that a mixed infection was certainly not present, because only large rings of the tropical parasites, situated in chromocytes showing Maurer's spots and crescent bodies, could be found.

In addition to the blood constituents already described, the forms shown in figs. 1, 2, and 3 were seen in the slides. These were examined with a Zeiss oil-immersion one-twelfth objective and No. 4 ocular, giving a magnification of 950. However, as an exact representation of all the details presented many difficulties when such an enlargement was used, I sketched figs. 1, 2, and 3 on a scale about 1.5 times greater, and they thus possibly represent an enlargement of 1,500; on the other hand, figs. 4 and 5 are magnified only 950 times. If, now, we examine figs. 1 and 2, it becomes evident that the parasitic body occupies almost all of the red blood corpuscle, which is neither enlarged nor faded. In fig. 3 it occupies approximately two-thirds of it; figs. 2 and 3 show some large parasites and fig. 1 two which are very minute. An elongated oval form may be seen in fig. 1. This is indented on the right margin and has an obtuse and slightly deflected point at the right upper corner, this point being quite similar to that of many young crescent bodies in the same blood slide. On the other hand, the opposite pole of these young gametes is either round or obtuse. In fig. 2 the parasite is oval, with the narrower pole directed downwards, whereas that of fig. 3 resembles a crescent body with a convex projection on the concave side. The protoplasm of the malarial parasite is stained a very light violet-blue in figs. 1, 2, and 3; in figs. 1 and 2 blue predominates; in fig. 3, violet-red, and this color of the protoplasm corresponds exactly to that of the crescent bodies in the blood slides. We find in this protoplasm in fig. 1, especially at the left-hand upper corner parallel to the contour of the parasite, but not touching the periphery, a distinct, band-like, light violet-red chromatic mass of basic material in which may be observed small, dark red-brown, chromatic bands which are generally situated at right angles to the former. In these small bands may be seen one or two chromosomes (nuclear masses) stained very dark purple. This light, red-violet basic substance is probably to be looked upon as a metamorphic change between the proper chromatic material and the protoplasm of the parasite. Between this broad, band-like basic mass and the periphery of the parasite we see imbedded in fig. 1 small, detached, chromatic points. In fig. 2 the light red-violet, band-like basic substance runs parallel to the periphery of the parasite, but touches the circumference almost at every point and incloses

three-fourths of the contour of the parasite. In the right, lower quarter there is an isolated islet of the same substance in which are imbedded two large, detached chromosomes.

Whereas we detect about 15 chromatic nuclear dots in fig. 1, in fig. 2 we find only about 12 of them. In fig. 3, where both poles and the remainder of the parasite show very distinctly that it is derived from a crescent body, the chromatic band may easily be distinguished as a crescent which runs from the upper left- to the lower right-hand corner of the parasitic body in such a manner that the greater part of the convex margin of this band does not touch its concave inner side. A protoplasmic zone, which in some places is very narrow, may be seen intact between the two margins. In the basic mass of this band we find imbedded darker chromatic dots. In the upper half these are very rare, are small and not very distinctly differentiated; in the lower half they are much more distinct and are more or less detached, resembling particles of the basic material. Indeed, two of these spots lie in the contiguous protoplasmic substance of the parasite. Furthermore, we find in fig. 1 an indefinite mass in the lower left-hand segment, consisting of irregularly scattered, varicolored, small chromatic dots, with transparent spots situated between them, making it appear as though the parasite were perforated by pinpricks. Between these, and also in the upper right-hand portion, a very typical, yellow-brown, coarse pigment occurs which bears all the peculiarities of the pigment of the crescent bodies. This spot, which is not easily defined, may, after the analogy of the tertian macrogamete, be considered as an unfertilized body (*Restkörper*), in course of decomposition. Two large perforations may clearly be seen in fig. 2, and one smaller one, somewhat beyond the actual center of the parasite. Around these perforations the beautiful, yellow-brown pigment of the crescent body is visible. However, a distinct unfertilized body (*Restkörper*) can not be discovered.

In fig. 3 the corresponding pigment lies in the center of the protoplasm. Here it is very beautifully imbedded and arranged like the stamens of the calix of a flower.

We may affirm, without fear of contradiction, from the morphological characteristics already described, that in figs. 1 and 2 we are dealing with segmenting forms. I wish by the following argument to meet the objection raised by Doctor Rodenwaldt that, after all, these might be microgametocytes, the chromatic substance of which is in the act of disintegration, the microgametes being about to be expelled.

1. As far as I know, microgametocytes, of which the chromatic substance had already been segmented for the microgametes which are to

be expelled, have never been seen in instantaneously congealed blood slides containing tropical gametes. A segmentation of the chromatic substance which has progressed as far as it is shown in figs. 1 and 2 would also, in my opinion, be impossible during the few seconds occupied by the process of congealing, unless we were inclined to concede that the segmentation had already begun in the circulating blood, which, however, has not up to the present time been proved. We can only continuously observe and follow the expulsion of the microgametes in fresh, uncongealed blood containing male gametes for fifteen to thirty minutes after its withdrawal from the body.

2. The chromatic particles, if they were destined for the microgametes which are about to be expelled, would greatly surpass the latter in number. In fig. 1 we have approximately 15 of these and in fig. 2 about 12. As far as I know, such a large number of microgametes is never formed by one microgametocyte.

3. The parasitic forms sketched in figs. 1 and 2 are much too large for tropical microgametocytes. Moreover, the form of this intracellular parasite is not in accord with such an hypothesis. It follows that we have to do with a sporular form. Therefore, we have only to decide, first, whether it is a case of a sexual segmentation or one of parthenogenesis of a macrogamete, and, second, the species of the parasite.

Forms of malarial parasites of other mammals and birds may be immediately excluded from consideration, because it has been proved by experiment that human blood can not be infected by these species. Hence we are limited in our differential diagnosis to the tertian, quartan, and tropical parasites. Figs. 1 and 2 present no point of similarity to the product of a quartan schizogenesis; the number of chromatic particles for the merozoites is much too great, the pigment is yellow-brown and coarse, the protoplasm light violet-red, instead of light azure-blue as it should be in the quartan parasite with Giemsa's stain. Moreover, after an exhaustive examination of the blood preparation, no sign of quartan infection in the form of rings, band-like parasites, or characteristic gametes can be found; finally, fig. 2 exhibits many and fig. 1 two small Maurer's spots.

When the red blood cells are infected by ring-formed parasites, they always exhibit Maurer's spots. Since I did not find a quartan infection during the examination of the blood, we may also exclude from our discussion the parthenogenesis of the female gamete, which until now has never been observed.

A tertian schizogenesis and a parthenogenesis of the tertian macrogamete are just as readily excluded because of the morphologic aspect

of the segmenting form, the staining reaction with respect to Giemsa's solution, the kind and color of the pigment, the absence of Schuffner's dots, the fact that the infected chromocytes are neither enlarged nor faded, and the lack of further tertian forms such as rings, amœbic forms, and gametes. In order to confirm the preceding view, namely, the inadmissibility of tertian parthenogenesis by comparison, I have added figs. 4 and 5, drawn with a magnification of 950 diameters. These specimens were prepared in an analogous manner and were stained with Giemsa's solution like the preparation represented by figs. 1, 2, and 3, with the difference that they were stained for a shorter time.

Fig. 4 demonstrates the first stage, where the light-blue protoplasm is separated very distinctly to the right and left. The former is the *Restkörper,* containing the expelled protoplasm destined to degenerate, a small chromatic mass, and a small quantity of pigment. The right side includes a spindle-shaped chromatic band, in which there may already distinctly be observed a differentiation in the form of the darker granules, for the subsequent segmentation of the chromosomes.

Fig. 5 represents the final stage of the parthenogenesis. The *Restkörper* is to be found situated in the upper left-hand corner with a certain amount of marginal, violet-red, chromatic substance. In the lower right-hand quadrant may be seen a light-blue sporular form, with eight chromatic particles destined for the young parasites. The *Restkörper* and segmenting form are very clearly discerned in both illustrations. Furthermore, a very marked enlargement and irregular, angular metamorphosis of the red blood cells may be distinguished in both of the figures. The cells exhibit beautiful examples of Schuffner's dots. Both forms of parthenogenesis were obtained from two of my blood slides made at Koeta-Radja two and a half years ago. These I submitted for verification to Von Prowazek, Mayer, Rodenwaldt, and Gonder, of the Institut für Schiffs und Tropen-Krankheiten at Hamburg. These observers confirmed my own conclusions.

It only remains to decide whether the forms illustrated in figs. 1 and 2 result from schizogenesis or parthenogenesis of the tropical parasite. I had an opportunity of comparing my blood slides with a long series of very characteristic schizogenesis of the tropical parasite at the Institut für Schiffs und Tropen-Krankheiten at Hamburg. The differences are very important and are as follows:

Figs. 1 and 2.	*Schizogenesis of the tropical parasite.*
(*a*) *Size.* The segmenting form fills almost the whole normal-sized red blood cell.	Segmenting forms reach a maximum of two-thirds of the size of the cell.
(*b*) *Shape.* Fig. 1: Elongated oval-shaped, with a slightly indented margin on the right and terminating in an obtuse and slightly bent point. Fig. 2: Oval with its center lying slightly outside the center of the blood cell.	Mostly circular and generally situated in or near the center of the red blood cell.
(*c*) *Chromosomes.* Large, coarse, purple-brown, peculiarly imbedded and arranged in a red-violet basic substance which is band-like in form and which extends in a curve parallel to the margin of the parasite.	Small, fine, purple, separated from one another, and distributed in a circular manner around the pigment which lies in the center.
(*d*) *Protoplasm.* Stained light blue-violet and identical with that of the crescent bodies.	Light azure-blue, including the small chromosomes more or less.
(*e*) *Pigment.* Coarse, yellow-brown, scattered, excentrically situated, quite similar to the pigment of the crescent bodies.	Fine, dark-brown or black, mostly concentrated in one clump, generally situated in the center and surrounded by the merozoites.

GENERAL CONCLUSIONS.

In view of the differences noted above, which have been investigated also by Nocht, Von Prowazek, Mayer, Rodenwaldt, and Gonder, I believe I can exclude the schizogenesis of the tropical parasite in the case in question, so that nothing remains but to admit that here we are dealing with parthenogenetic forms of the tropical macrogamete. As confirmatory evidence, I desire to emphasize the following facts:

1. The size and shape of the segmenting parasites, in which may be observed very distinctly in the upper right-hand corner of fig. 1, the obtuse, slightly bent point, the crescent body, which point may be seen much better in fig. 3, where the two poles can not be mistaken.

2. The light, blue-violet staining reaction of the protoplasm of the parasite, identical with that of the crescent bodies in the same blood preparation and entirely different from the light azure-blue coloring of the quartan and tertian parasites, which have been treated in the same way.

3. The coarse structure, size, and peculiar arrangement of the chromosomes, which for the greater part lie at right angles to the red-violet band-like basic substance.

4. The coarse, yellow-brown pigment distributed in a small group, for the most part excentrically situated and quite similar to that of the crescent bodies.

5. The absence of a mixed infection, so that only a simple tropical infection can be present.

Whereas fig. 4, with its spindle-shaped chromatic band, presents the initial stage of the parthenogenesis of the tertian macrogamete, and fig. 5, the completed segmentation, I wish to call attention to fig. 3, which shows the band-like chromatic mass in which an early division is already to be observed, analogous to the process illustrated in fig. 4. This form I consider as the earlier stage of the parthenogenesis of the tropical macrogamete, whereas figs. 1 and 2 represent the almost finished sporulating stages of the same metamorphosis. Moreover, fig. 1 in the lower left-hand quarter probably shows the *Restkörper*.

I do not conceal the fact that the form illustrated in fig. 3 was considered at Hamburg to resemble a young crescent body; nevertheless, I consider myself bound to adhere to the opinion which I have set forth in this paper, an opinion which has been arrived at only after a careful and exact comparison of this form with the young crescent bodies in the same blood slide, which latter have a totally different appearance.

ILLUSTRATIONS.

PLATE I.

FIGS. 1 to 3. Parthenogenesis of the tropical macrogamete ± 1,500 x.
FIGS. 4 and 5. Parthenogenesis of the tertian macrogamete ± 950 x.

Fig. 1.

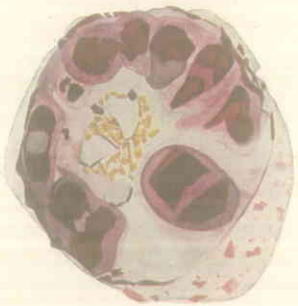

Fig. 2.

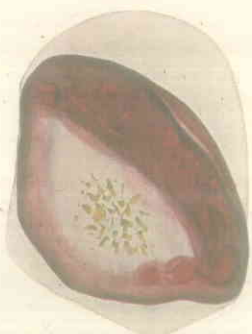

Fig. 3.

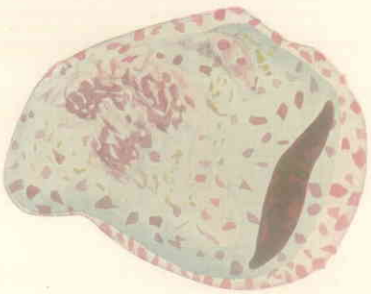

Fig. 4.

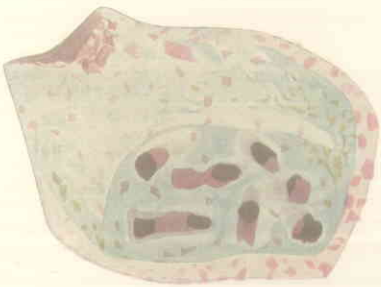

Fig. 5.

PLATE I.

ON MALARIA PARASITES OF THE ORANG-OUTAN.

By G. SHIBAYAMA.[2]

(From the Institute for Infectious Diseases, Tokio.)

The first and only investigation of the malarial parasite in the orang-outan was made by Halberstaedter and Prowazek in Java in the year 1907, while reports on the malarial plasmodium of *Maccacus* are not lacking in the literature. By a comparative study, the authors established the species diagnosis between the parasite of *Maccacus* and that of the orang-outan.

The plasmodium of the orang-outan which I have studied and which is illustrated in the accompanying plates does not differ from the *Plasmodium petheci* spec. nov. Halb. u. Prow., except in the absence of stippling (*"Zipfelung"*) of the red blood corpuscles. The young forms appear as small rings which, as in the case of the tropical parasite of man, are composed of a nucleus staining red and of a crescent-like mass of protoplasm staining blue. Ribbon-like forms resembling those of the quartan parasite of man are also observed. In the adult parasite a vacuole appears between the nucleus and the protoplasm, and rod-like pigment granules are also present. The chromatin of the nucleus enlarges and becomes differentiated as the parasite increases in size.

The sexual forms resemble the quartan parasite in respect to pigmentation and staining reactions. The male sexual forms are rich in chromatin. The protoplasm takes a paler stain and is but slightly pigmented. The female gametocytes on the contrary contain dark protoplasm and are rich in pigment. The schizogony of the parasite of the orang-outan takes place after the manner of the human tertian parasite. The nucleus of the schizonts divides into 12 to 16 parts, most of the pigment being situated in the center of the cell.

[1] Read at the first biennial meeting of the Far Eastern Association of Tropical Medicine, held at Manila, March 12, 1910. Translated from the German.

[2] Delegate from His Imperial Japanese Majesty's Government.

ILLUSTRATIONS.

PLATE I.

FIG. 1. *Plasmodium petheci sp. nov.* Ringform like the tropical plasmodium.
2. Amœboid form with vacuole.
3. Double infection.
4. Adult form with pigment granules and differentiated chromatin.
5. Microgametes like quartan parasites.
6. Macrogametes like quartan parasites.
7. Schizogony according to the type of the human tertian parasite.

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 6.

FIG. 7.

PLATE I.

MALARIAL FEVER DURING THE PUERPERIUM.[1]

By J. M. Atkinson.[2]

When considering the subject-matter for a paper to be read before this most important gathering, containing, as it does, members from practically all the countries east of Suez, it occurred to me that some useful practical deductions might be arrived at by the discussion of the puerperium complicated by malaria. As a text for these remarks I shall give you the notes of two of the cases which have occurred in my twenty-two years of clinical experience in Hongkong.

The first was that of an English lady, who was admitted to the Maternity Hospital of the Government Civil Hospital on December 21, 1902. The patient was a primipara at full term, who stated that she had been suffering from fever for some days before admission, and that her temperature had been as high as 40.5°C. (105°F.). Though not in labor, the patient was recommended to enter the hospital at once, both on account of the fever and especially because both she and her relatives were very anxious in regard to her condition. She was admitted the same evening, and her temperature at 9 p. m. was 38.9°C. (101.6°F.). She was placed on low diet, and a diaphoretic mixture to be taken every four hours was prescribed. On the following morning her temperature was normal.

A blood film was examined at this time and simple *tertian* parasites and *ring-forms* were found. Three-tenths of a gram (5 grains) of quinine was ordered every four hours. At 6 p. m. on the evening of the 22d labor pains commenced. At 6 a. m. on the 23d her temperature was 37.8°C. (100°F.), and at 12.15 p. m. the child was born.

Beyond slight post-partum hæmorrhage, which was checked with a hot lysol douche, the labor was normal.

Quinine was continued as before. At 8 p. m. her temperature was 40°C. (104°F.). The fever was evidently due to a tertian attack, as next morning the temperature was normal. From this date there was no more fever; the quinine was continued, and on the 27th the note was made that a blood film showed some *ring-forms* but no *simple tertian* parasites.

The patient was discharged eighteen days afterwards, free from malaria, repeated blood examinations having shown no parasites.

This case is one of interest from the fact that the patient had only arrived in the colony a few months previously. She had never to her knowledge had malaria before and did not come from a malarial country.

[1] Read at the first meeting of the Far Eastern Association of Tropical Medicine, held at Manila, March 10, 1910.

[2] Principal medical officer, Hongkong; delegate from the government of Hongkong.

During her stay in Hongkong, however, she had resided in a locality (MacDonald Road) known to be infested with *Anopheles* and where malarial fever was rife. In the previous summer extensive building operations had been carried on in this district of the town, many European houses having been built there, and malarial fever was very prevalent. In the Annual Report of the Medical Department for the year 1902[3] I find, under the heading "Antimalarial measures," the following:

> This locality had been especially dealt with, much undergrowth had been removed, pools of stagnant waters had been drained, and the *nullahs* partially trained. In the winter months a general fumigation of the servants' quarters in the houses of this district was carried out by the sanitary board staff with the object of killing off the *Anopheles* and their ova.

In this case immediate blood examination revealed the cause of the fever, and the subsequent rise of temperature after labor caused no anxiety as to sepsis. The blood examination also enabled the attending physician to ease the mind of the patient and assure her relatives as to the favorable prognosis of the case.

The history of the next case is one in which an attack of malarial fever induced premature labor. It occurred as recently as November last and the history is briefly as follows:

> On November 27 last I received a telephone message that a case of premature labor was being sent to the Victoria Hospital for Women and Children from Stonecutters' Island.
>
> I must mention that arrangements have been made whereby the women and children from the military, when sick, are sent to this hospital.
>
> At 3.45 p. m. that day I received a note from the sister on duty stating that "the patient had just arrived, looking very ill, her temperature being 40°C. (104°F.) and pulse 128. She complained of severe headache, great thirst, and had severe pain in the right side. The perineum was ruptured, very much swollen, and tender to the touch."
>
> I sent word that the patient should be admitted to the isolation ward, as the hospital was rather full, there being several recent confinement cases under treatment. It appeared to me that the case might be one of acute puerperal sepsis.
>
> On examination I found the patient to be an English woman with a small, eight-months' baby, weighing but 2 kilos (4¼ pounds). Her condition was as described by the sister; in addition, she had a brown, dry tongue and was complaining of severe headache.
>
> She was the wife of a sergeant in the Royal Garrison Artillery and had been in Hongkong for two years, during the whole of which time she had been living in the married quarters at Stonecutters' Island, situated in the harbor some 3 miles from Victoria.
>
> Eighteen months before she had suffered from an eight weeks' abortion, otherwise her medical history was good and she had not previously had malarial fever.
>
> As she was so ill I did not disturb her further that night, but gave her 1 gram (15 grains) of trional and ordered fomentations of corrosive sublimate to be applied to the swollen, torn perineum. The following morning the patient had

[3] Report of the Principal Civil Medical Officer for the year 1902, p. 264.

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[3] Report of the Principal Civil Medical Officer for the year 1902, p. 264.

improved; she had slept well, the headache was gone, and her temperature and pulse were normal. The swelling of the perineum was much less marked and the lochia were not offensive.

I then questioned her as to her illness and she informed me that the fever had commenced suddenly with an attack of shivering on the 25th of the month. On the 26th labor pains set in, and the child was born at 3 a. m. on the 27th. A military doctor had seen her and immediately ordered her removal to Hongkong.

This necessitated her traveling by a launch for over an hour, and then being transferred on a stretcher to the Victoria Hospital, some 1,000 feet above the sea level. Naturally, she arrived in a somewhat collapsed condition, but soon rallied on being placed in a warm bed and after being given some stimulant. The pain she described in her right side was evidently muscular.

On examining her blood on the morning of the 28th, I found it to be swarming with benign tertian parasites. She was given 0.3 gram (5 grains) of quinine every four hours, together with an effervescing mixture containing 4 minims of dilute hydrocyanic acid to help her to retain the drug. As the swelling of the perineum had subsided, I removed several small sloughs and inserted a deep silkworm suture under cocaine anæsthesia. Her temperature did not rise again to 37.8°C. (100°F.), and she made an uninterrupted recovery. On January 3 she was discharged from the hospital quite well, accompanied by her baby, who then weighed 3 kilos (6¾ pounds).

Both these cases might have caused considerable anxiety to the medical attendant had they not occured in a malarial country where the physician was alert for this complication. So much does malaria complicate practically all illnesses in Hongkong, especially in the hot summer months, that it is a rule with us to give parturient cases quinine whenever there is the smallest rise of temperature after labor. The practical question must also be considered of how far one is justified in giving quinine daily as a prophylactic to women during the period of pregnancy. Many physicians are chary of doing so, as they think it may from its ecbolic action on the uterus induce abortion or premature labor. In my opinion, it is malarial fever that is more likely to produce that effect, and I am in the habit of always advising the daily dose of 0.130 gram (2 grains) during the summer months, especially to pregnant women whom I have known to have been subject to attacks of malarial fever.

The following is quoted from Dr. Albert H. Smith:[4]

> Quinine has no power in itself to excite uterine contractions, but simply acts as a general stimulant and promoter of vital energy and functional activity. In normal labors at full term, its administration in a dose of 15 grains is usually followed in as many minutes by a decided increase in the force and frequency of the uterine contractions, changing in some instances a tedious exhausting labor into one of rapid energy, advancing to an early completion.

Quinine promotes the permanent tonic contraction of the uterus, after the expulsion of the placenta. Women that had flooded in former labors escaped entirely, there not having been one instance of post-

[4] *Trans. Coll. Phys. Philadelphia* (1875), 183.

partum hæmorrhage in 42 cases so treated. It also diminished the lochial flow where it had been excessive in former labors, the change being remarked upon by the patients; it consequently lessens the severity of the after pains. Cinchonism is very rarely observed as an effect of large doses in parturient women.

I have discussed this question of the supposed ecbolic physiological action of quinine with several of the medical practitioners in Hongkong and they agree with me that this action is very slight; some, in concurrence with myself, doubt that it has any effect in this way, and I am inclined to believe with Dr. Albert Smith that it acts more as a general stimulant and promoter of vital energy and functional activity. Of one thing I am convinced, and that is that when quinine is administered in malarial fever it expends its energy in killing the plasmodium and does not produce any deleterious effects on the system.

The large doses of quinine which are sometimes required seem extraordinary, but these doses can be given with impunity in some of those malignant cases of malarial fever which every physician who practices in malarial countries has occasionally to deal with.

Similarly, large doses of other drugs are sometimes necessary in certain other diseases and are tolerated by the economy. I need only instance mercury in the treatment of syphilis and arsenic in the treatment of certain forms of anæmia.

TROPICAL BRONCHOMYCOSIS. OBSERVATIONS ON A NEW SPECIES OF EPIDERMOPHYTON FOUND IN TINEA CRURIS. A NEW INTESTINAL SPIRILLUM.[1]

By ALDO CASTELLANI.[2]

TROPICAL BRONCHOMYCOSIS.

During the six years of my residence in Ceylon I have often been struck by the comparatively large number of cases of subchronic and chronic bronchitis which the physician on superficial clinical examination would suspect to be of a tubercular nature, while complete investigation shows constant absence of tubercle bacilli in the sputum. The ophthalmic and cutaneous reactions are negative, and inoculations of the sputum into susceptible animals are also negative. Some of these proved to be cases of bronchospirochætosis, the condition described by me in 1905. In other instances, however, neither spirochætæ nor tubercle bacilli are found, and in a certain number fungi more highly organized than bacteria are present. These are cases of bronchomycosis. I have encountered at least 20 cases of this affection in Ceylon, basing the diagnosis on the result of the microscopic examination, and have culturally studied the fungi isolated from 4 cases. In Ceylon a mild and a graver type of this disease may be distiguished. In the mild type the patient complains of cough with mucopurulent expectoration. There is no fever, the general condition is fairly good; the physical examination of the chest will show nothing at all, or a few moist and dry râles. In the severe type, the disease closely resembles phthisis, the patient becomes emaciated, there is hectic fever, mucopurulent and bloody expectoration; the physical examination of the chest reveals patches of dullness, fine crepitations and pleural rubbing. I may cite a few instances of each type.

Case 1 (mild type).—Mr. B. A., young European, occupation tea-taster, has been in Ceylon eight years, has had no disease of importance. He remains several

[1] Read at the first biennial meeting of the Far Eastern Association of Tropical Medicine, held at Manila, March 12, 1910.

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hours daily in rooms full of tea dust and fluff and says that he often snuffs various teas, putting a pinch of the tea in his nostrils. For six months he has complained of a cough with a certain amount of mucopurulent expectoration. No fever. The examination of the chest reveals nothing. Microscopic examination of the sputum for tubercle bacilli is negative. In fresh preparations a few branching mycelial tubes are visible; very few free spores.

Treatment: Ordinary cough mixtures were useless; potassium iodide in large doses cured the condition in four weeks.

Case 2 (mild type).—Mr. S. A., European planter; is compelled to remain for a couple of hours daily in the tea factory. He consulted me in September, 1909, and informed me that he was suffering from what the planters call "tea-factory cough." He had mucopurulent expectoration; no fever; his general condition was fairly good. The microscopic examination of the sputum for tubercle bacilli was negative. Numerous spores and mycelial tubes of an oïdium-like fungus were present.

Treatment: Potassium iodide in 1.0 gram (15 grains) doses four times daily cured him in three weeks.

Case 3 (severe type).—Mr. M., a planter. The sputum had been examined many times for tubercle bacilli by his medical attendants with negative results. When the patient arrived in Colombo he was extremely weak. He had serotine fever, bloody expectoration, fine crepitations and pleural rubbing on both sides. I examined the sputum many times for tubercle bacilli with negative results. Inoculation into guinea pigs was negative. Fresh preparations always showed numerous spores and some mycelial threads. Cultures were made from the sputum on agar and on various sugar media. The only germ grown was a hyphomycete which showed the same morphological characters as that present in the fresh preparation, and a streptococcus. The patient became gradually worse. The cough was not relieved by guaiacol, duotol, nor by potassium iodide. He died three weeks after arrival.

Case 4.—Singhalese convict in Mahara jail, near Colombo. The symptoms of his disease were at first obscure, and various diagnoses, including typhoid, malaria, and phthisis, were suggested. The sputum was sent to me several times, but tubercle bacilli were always absent. Instead, a saccharomyces and oïdium-like fungus were found. The patient was later transferred to the clinic for tropical medicine where I kept him under observation for two months. The cough slowly decreased and finally stopped, the fever completely disappeared. His blood by means of Widal's reaction was shown to contain agglutinins for the cultures of the oïdium-like fungus, and the saccharomyces.

BACTERIOLOGICAL INVESTIGATION OF THE FUNGI FOUND IN THE FOUR CASES.

In four cases I plated from the expectorations and grew the hyphomycetes. In the case from the Mahara jail, as I briefly stated, two fungi were present—a saccharomyces and an oïdium—in the other three cases only oïdium-like fungi were observed.

Description of the saccharomyces.—In fresh preparations of the sputum the organism appeared as oval, rounded budding cells. It was Gram positive. The cultural characteristics are summarized in the following table.

The cultural characters of the saccharomyces at 37°C.

Medium.	Per cent.	Characters.
Glucose-litmus broth	2	Acid and gas. Thick pellicle.
Lævulose-litmus broth	1	Do.
Maltose-litmus broth	2	Practically no change. Thick white pellicle.
Galactose-litmus broth	1	Do.
Saccharose-litmus broth	2	Do.
Lactose-litmus broth	2	Do.
Mannite-litmus broth	2	Do.
Dulcite-litmus broth	2	Do.
Dextrin-litmus broth	1	Do.
Raffinose-litmus broth	1	Do.
Arabinose-litmus broth	1	No change. Delicate pellicle.
Nutrose-litmus broth	1	No change. Very delicate pellicle.
Inulin-litmus broth	1	Practically no change. Thick white pellicle.
Adonite-litmus broth	1	No change. Delicate pellicle.
Litmus milk		No change.
Broth		Clear, thin pellicle. Slight sediment at bottom of tube.
Peptone-water		Clear, slight sediment at bottom of tube. Practically no growth.
Serum		White growth. Not liquefied.
Agar		Delicate whitish growth. (Delicate on saccharose and acid maltose-agar.)
Glucose-agar		Very thick growth.

Description of the oidium-like fungi.—In fresh preparations of sputum, septate mycelial tubes 3 to 4 μ in breadth were seen at the terminal end of each, of which two to four shorter ovoid elements could often be observed; numerous free, oval, roundish spores 4 to 8 μ were also seen. The organism was positive for Gram's stain. On ordinary agar and various sugar agars the fungi grew abundantly, producing roundish, thick, white, creamy colonies which later coalesce. The cultural characteristics in various sugar media are collected in the following table, in which for comparative purposes those of the saccharomyces are repeated.

The cultural characters of oïdium (strain 1) at 37°C.

Medium.	Per cent.	Characters.
Glucose-litmus broth	2	Acid and gas. No pellicle. Abundant growth at bottom of tube.
Lævulose-litmus broth	1	Do.
Maltose-litmus broth		Acid. Gas in 2 days. No pellicle. Abundant growth at bottom of tube.
Galactose-litmus broth	1	Acid and very slight gas. No pellicle. Fairly abundant growth at bottom of tube.
Saccharose-litmus broth	2	Acid and gas. No pellicle. Abundant growth at bottom of tube.
Lactose-litmus broth	2	No change. Very slight growth at bottom of tube.
Mannite-litmus broth	2	Do.
Dulcite-litmus broth	2	Do.
Dextrin-litmus broth	1	No change. Fairly abundant growth at bottom of tube.
Raffinose-litmus broth	1	No change. Very slight growth at bottom of tube.
Arabinose-litmus broth	1	No change. Fairly abundant growth at bottom of tube.
Nutrose-litmus broth	1	No change. Slight growth at bottom of tube.
Inulin-litmus broth	1	No change. Very slight growth at bottom of tube.
Adonite-litmus broth	1	No change. Fair growth at bottom of tube.
Litmus milk		No change.
Broth		Clear. Fine (thin) pellicle. Slight sediment.
Peptone water		Clear. Thin pellicle. Slight sediment.
Serum		Creamy growth, surrounded by a zone of yellowish-pink color. Not liquefied.
Agar		Thin white moist growth.
Glucose-agar		Very thick wax-like growth. (Also very thick on saccharose and acid maltose-agar.)

The cultural characters of oïdium (strain 2) at 37°C.

Medium.	Per cent.	Characters.
Glucose-litmus broth	2	Acid and gas. No pellicle. Good growth.
Lævulose-litmus broth	1	Do.
Maltose-litmus broth	2	Do.
Galactose-litmus broth	1	Acid and gas. No pellicle. Fair growth.
Saccharose-litmus broth	2	Acid and gas. No pellicle. Good growth.
Lactose-litmus broth	2	No change. Slight growth at bottom of tube.
Mannite-litmus broth	2	Do.
Dulcite-litmus broth	2	Do.
Dextrin-litmus broth	1	No change. Fair growth at bottom of tube.
Raffinose-litmus broth	1	Do.
Arabinose-litmus broth	1	Do.
Nutrose-litmus broth	1	Do.
Inulin-litmus broth	1	Do.
Adonite-litmus broth	1	Do.
Litmus milk		No change.
Broth		Clear. Very slight pellicle. Practically no growth.
Peptone water		Clear. Practically no growth.
Serum		Growth white and shining with the serum under the growth and immediately surrounding it of a distinct reddish color. Not liquefied.
Agar		Very thin white growth.
Glucose-agar		Thick growth. White waxy surface. (Also very thick on saccharose and acid maltose-agar.)

The cultural characters of oïdium (strain 3) at 37°C.

Medium.	Per cent.	Characters.
Glucose-litmus broth	2	Acid and gas. No pellicle. Good growth at bottom of tube.
Lævulose-litmus broth	1	Do.
Maltose-litmus broth	2	Do.
Galactose-litmus broth	1	Acid in 5 days. No gas.
Saccharose-litmus broth	2	Acid and gas in 5 days.
Lactose-litmus broth	2	No change. Very slight growth at bottom of tube.
Mannite-litmus broth	2	Do.
Dulcite-litmus broth	2	Do.
Dextrin-litmus broth	1	Do.
Raffinose-litmus broth	1	Do.
Arabinose-litmus broth	1	Do.
Nutrose-litmus broth	1	Do.
Inulin-litmus broth	1	Do.
Adonite-litmus broth	1	Do.
Litmus milk	----------	No change.
Broth	----------	Clear. Slight sediment. Practically no growth.
Peptone water	----------	Do.
Serum	----------	White growth. Slight pigmentation surrounding bottom of growth after 10 days. Not liquefied.
Agar	----------	Very thin white growth.
Glucose-agar	----------	Thick white wax-like growth. (Also very thick on saccharose and acid maltose-agar.)

The cultural characters of oïdium (strain 4) at 37°C.

Medium.	Per cent.	Characters.
Glucose-litmus broth	2	Acid and gas. No pellicle. Good growth at bottom of tube.
Lævulose-litmus broth	1	Do.
Maltose-litmus broth	2	Do.
Galactose-litmus broth	1	Acid. No gas. No pellicle. Good growth at bottom of tube.
Saccharose-litmus broth	2	Acid. No pellicle. Good growth at bottom of tube. Slight gas at eleventh day.
Lactose-litmus broth	2	No change. Good growth at bottom of tube.
Mannite-litmus broth	2	Do.
Dulcite-litmus broth	2	Do.
Dextrin-litmus broth	1	Do.
Raffinose-litmus broth	1	Do.
Arabinose-litmus broth	1	Do.
Nutrose-litmus broth	1	Do.
Inulin-litmus broth	1	Do.
Adonite-litmus broth	1	Do.
Litmus milk	----------	No change.
Broth	----------	Clear. Slight sediment. Practically no growth.
Peptone water	----------	Do.
Serum	----------	Whitish growth. Not liquefied.
Agar	----------	Thin white moist growth.
Glucose-agar	----------	Very thick wax-like growth. White. (Also very thick on saccharose and acid maltose-agar.)

Cultural reactions at 37°C. after fourteen days.

[A.=production of acid; G.=production of gas; O=no change.]

	Glucose.	Lævulose.	Maltose.	Galactose.	Saccharose.	Lactose.	Mannite.	Dulcite.	Dextrine.	Raffinose.	Arabinose.	Nutrose.	Inulin.	Adonite.	Litmus milk.	Broth.	Peptone water.	Liquefaction of serum.	Liquefaction of gelatine.	Indole.
Oïdium strain I	A. G.	A. G.	A. G.	A. G.[a]	A. G.	0	0	0	0	0	0	0	0	0	0	[b]0	[b]0	0	0	0
Oïdium strain II	A. G.	A. G.	A. G.	A. G.	A. G.	0	0	0	0	0	0	0	0	0	0	[b]0	[b]0	0	0	0
Oïdium strain III	A. G.	A. G.	A. G.	A.	A. G.	0	0	0	0	0	0	0	0	0	0	[c]0	[c]0	0	0	0
Oïdium strain IV	A. G.	A. G.	A. G.	A.	A.	0	0	0	0	0	0	0	0	0	0	[c]0	[c]0	0	0	0
Saccharomyces	A. G.	A. G.	0	0	0	0	0	0	0	0	0	0	0	0	0	[b]0	[c]0	0	0	0

[a] Very slight gas. [b] Thin pellicle. [c] Practically no growth.

It will be seen from the table that the four strains of oïdium isolated are identical in all their cultural characteristics except that numbers 1 and 2 produce acid and gas in galactose, while strains 3 and 4 produce only acid. This has always remained constant, although I have repeated the reactions several times. All the four strains are different from the ordinary *Oïdium albicans* and *Oïdium lactis*, as they have no action on milk, at least within three weeks. As regards the saccharomyces, its cultural characteristics are different, as far as I know, from any other saccharomyces as yet described.

CONCLUSIONS.

1. A type of bronchomycosis in which oïdium-like and saccharomyces-like fungi are found is not rare in Ceylon. The condition might be called bronchooïdiomycosis, or more briefly bronchooïdiosis.

2. Two types of the condition may be clinically distinguished, a mild and a severe one; the latter closely resemblies phthisis. The mild type is apparently amenable to treatment with potassium iodide.

3. The strains of oïdia found in my cases are different from the ordinary *Oïdium albicans* and *Oïdium lactis*, as they do not affect milk.

4. All the strains found by me are identical in all respects, except that some produce gas in galactose and others do not. For the oïdium which produce gas in galactose I propose the name *Oïdium tropicale;* for the saccharomyces I suggest the name *Saccharomyces krusei.*

5. The diagnosis of bronchoöidiosis can be made only by bacteriologic methods. It is differentiated from phthisis by the absence of tubercle bacilli and the negative animal inoculations; from bronchial spirochætosis by the absence of spirochætæ; and from endemic hæmoptysis by the absence of the ova of the trematode.

6. Care should be taken before making the diagnosis of bronchomycosis that the sputum is collected in sterile vessels and examined as soon as possible, because sputa left exposed to the air frequently become contaminated in the Tropics with various species of nonpathogenic saccharomyces and oïdia. Primary bronchomycosis should be also differentiated from those cases of chronic debilitating disease in which *Oïdium albicans* spreads from the mouth to the bronchi.

OBSERVATIONS ON A NEW SPECIES OF EPIDERMOPHYTON FOUND IN TINEA CRURIS.

In 1905 I separated dhobie itch from the ordinary forms of tinea corporis and Macleod suggested for the affection the term tinea cruris. I stated at the time that the eruption was caused by different species of fungi, the commonest of which a few months later I termed *Trichophyton cruris*. Pernet found and described a fungus from a case of tinea cruris, and later I gave the name of *Tr. perneti* to this species. In 1907 Sabouraud made an investigation into an epidemic of tinea cruris occurring in France and he also came to the conclusion that tinea cruris, or, as he prefers to call it, tinea inguinalis, should be separated from tinea corporis. He created a new genus for the trychophyton-like organism observed in the disease—*Epidermophyton*. The principal characteristics of this genus are that the fungi do not attack the hair follicles, do not produce suppuration, and that cultures show forms of degeneration in a very short time. Sabouraud isolated only one species in all of his cases; but in the Tropics, in my experience, there can not be any doubt of the plurality of species of the fungi producing tinea cruris or dhobie itch. The fungi described so far are:

1. *Epidermophyton cruris* Castellani, 1905. *E. inguinale* (Sabouraud), 1907. Colonies in maltose agar whitish, occasionally orbicular; later showing a greenish color.

2. *Epidermophyton perneti* Castellani, 1907. *Trychophyton perneti.* Colonies in maltose agar of a delicate pinkish color. The pinkish color is lost in subcultures.

To these two species I am now in position to add a third, which I will designate as *E. rubrum* and which I have isolated from two cases of the

so-called eczematoid type of tinea cruris. In one patient the eruption was localized to the groins, scrotum and thighs; in the other, besides the scrotum and thighs the armpits and portions of the chest and abdomen were affected. I may here remark that tinea cruris is not always localized in the groins or the armpits. The affection generally begins in these regions, but in many cases it may spread to any other part of the body except the scalp; indeed I have seen a few cases in which the disease started on the chest, arms, or face, and then spread to the groin and armpits.

DESCRIPTION OF THE FUNGUS.

In preparations from the affected parts in potassium hydroxide mycelial tubes and free spores are observed, identical to those seen in *E. cruris* and *E. perneti*, and similar to those of any *Trychophyton* of the megalosporon type. The spores are large, globular, 4 to 6 μ in diameter, with a double contour. The mycelial tubes, 2 to 3.5 μ, are straight, bent, or variously shaped.

CULTURES.

Sabouraud agar.—The growth begins to appear four to six days after inoculation as a raised, red spot which gradually enlarges. The full-grown colonies are of a deep red color, either with a central knob or crater form, and are partly covered, especially the central knob, by a white down. In old cultures this may cover the entire growth and may hide the red pigmentation almost completely. The pigmentation even as far as the nineteenth subculture has not been lost.

Glucose agar.—The cultures are of a very deep blood-red color, and portions of the medium take the same tint. In old cultures a large amount of white down is present over the entire surface of the growth, but scraping this out, the red pigmentation is then extremely well marked. The red pigmentation at the time of writing is still characteristic in the nineteenth transplanted subculture.

Mannite.—Colonies deep red, covered with white down and with central knob. After a time the whole surface of the growth shows abundant, whitish fluff.

Maltose.—In 2 per cent maltose agar, alkaline or acid, the colonies are whitish; in 4 per cent maltose agar they may occasionally be red.

Ordinary agar.—The fungus grows well, white colonies with a central knob being formed; later on these show a peripheral greenish ring, encircled by a thin whitish or whitish-green zone.

Saccharose agar.—A central white knob and later a yellowish ring are green, finally a whitish zone forms at the periphery.

Glycerine agar.—White growth with central white knob and white powdery surface.

The principal cultural characteristics already described and those presented in other media are collected in the following table.

The principal cultural characters of epidermophyton rubrum.

Medium.	Per cent.	Characters.
Sabouraud agar		Red growth.
Glucose agar	2	Deep red growth with later white fluffy surface.
Mannite agar	2	Do.
Saccharose agar	2	Cream growth with white, powder-like surface.
Maltose acid agar	2	Do.
Maltose alkali agar	2	Do.
Lactose agar	2	Do.
Glycerine agar	2	Do.
Saccharine agar	2	White growth with white, powder like surface. (After 1 month the growth on saccharose presented a white central knob surrounded by a grayish-white zone. This grayish-white zone was surrounded by a distinct yellow ring and outside this was another zone of gray white color.)
Agar (plain)		As saccharine.
Gelatine		White growth. Nonliquefied after 21 days.
Serum		Nonliquefied after 21 days.
Litmus milk		Alkaline. Complete separation in 7 days.
Broth		Growth on surface and at bottom of tube of pale yellow color.
Peptone water		Growth at bottom of tube. White.
Lactose-litmus broth	2	No acidity. Good surface growth of greenish-white color.
Mannite-litmus broth	2	No acidity. Good surface growth. White.
Dulcite-litmus broth	2	No acidity. Good growth at bottom of tube.
Glucose-litmus broth	2	No acidity. Good surface growth. Red with whitish down.
Maltose-litmus broth	2	No acidity. Superficial whitish growth.
Saccharose-litmus broth	2	No acidity. Superficial greenish-white growth.
Lævulose-litmus broth	2	No acidity. Abundant greenish-white surface growth.
Inulin-litmus broth	2	No acidity. Abundant greenish-yellow surface growth.
Raffinose-litmus broth	2	No acidity. Abundant greenish-white surface growth.
Galactose-litmus broth	2	No acidity. Abundant greenish-yellow surface growth.
Dextrin-litmus broth	2	No acidity. Only slight growth at bottom of tube, none on surface.
Arabinose-litmus broth	2	No acidity. Greenish-white surface growth.
Adonite-litmus broth	2	No acidity. Growth only at bottom of tube and very slight.
Nutrose-litmus broth	2	No acidity. Growth at bottom of tube and also abundant surface growth of greenish-yellow color.

Observations on subcultures.—Subcultures from a Sabouraud or glucose culture on Sabouraud or glucose media are deep red, like the original cultures, but subcultures on agar, saccharine agar, and saccharose agar are white, greenish or yellowish. Subcultures from an agar culture (white) or ordinary agar, saccharine, and saccharose are white, but if Sabouraud agar tubes or glucose or mannite

tubes be inoculated from a white agar colony, the fungus will not be white, but deep red. The development of the color of the fungus is therefore dependent upon the composition of the medium on which it is inoculated. In Sabouraud and mannite, and best of all in glucose media, the fungus so far as I have observed is red. At the present time I have the tenth generation and the fungus has the same characteristics as in the first.

Hanging drop cultures.—Hanging drop cultures in Sabouraud maltose or ordinary broth present the characteristics of the other *epidermophytons*. Reproduction takes place by budding and branching of the mycelial tubes; clamydospores are present. It is interesting to note the rarity of lateral conidia.

CONCLUSIONS.

1. Tinea cruris or dhobie itch is caused by several species of *Epidermophyton*.

2. The *Epidermophyton* I have described above may be added to the two already known, *Epidermophyton cruris* Castellani and *E. perneti* Castellani. For this new species I propose the name *E. rubrum*.

3. *Epidermophyton rubrum* is characterized principally by the deep red pigmentation of its growth in glucose, Sabouraud, and mannite agars; whereas it is white in ordinary agar. The pigmentation is remarkably persistent and continues to be quite characteristic at the time of writing (February, 1910), although the fungus has already been transplanted nineteen times in subculture.

A NEW INTESTINAL SPIRILLUM.

I have recently encountered two cases of a peculiar type of acute, fatal enterocolitis showing intermediate symptoms between dysentery and cholera. Some of the stools were serous and cholera-like; others consisted practically of mucus only. There was no blood. Both cases died within forty-eight hours. In one, the disease was said to have begun after eating dried fish. The stools, collected in sterile Petri dishes, were examined for *Vibrio choleræ*, with negative results. On the other hand, 85 per cent of the colonies which developed on bile-salt agar and ordinary agar were of a peculiar spirillum; the others resembled organisms of the colon group.

DESCRIPTION OF THE SPIRILLUM.

Morphology.—The spirillum varies greatly in length and in shape; the same preparation from an agar or broth culture will show some individuals 20 to 40 μ in length with 2 to 4 coils, and also short bacillary or comma-like forms. The difference in shape is so great that at first I believed I had encountered an instance of symbiosis between a bacillus and a spirillum, but even by plating and replating I never succeeded in separating the two forms, and therefore I consider them to be of one and the same organism. The spirillum is readily stained with the usual aniline dyes. It is Gram negative.

Cultural characteristics.—The cultural characteristics are given in the following table.

Cultural characteristics of the spirillum.

Medium.	Per cent.	Characters.
Litmus milk		No acidity. (After 8 weeks the milk was distinctly alkaline and peptonized.)
Broth		General turbidity; pellicle.
Peptone water		General turbidity; slight pellicle
Gelatine		Not liquefied.
Serum		Do.
Agar		Whitish. Coli-like.
Lactose-litmus broth	2	No change. Fair growth.
Saccharose-litmus broth	2	Do.
Dulcite-litmus broth	2	Do.
Mannite-litmus broth	2	Do.
Glucose-litmus broth	2	Do.
Maltose-litmus broth	2	Do.
Dextrin-litmus broth	1	Do.
Raffinose-litmus broth	1	Do.
Arabinose-litmus broth	1	Do.
Adonite-litmus broth	1	Do.
Inulin-litmus broth	1	Do.
Nutrose-litmus broth	1	Do.
Galactose-litmus broth	1	Do.
Lævulose-litmus broth	1	Do.
Indol		Negative.
Gram		Negative. Very motile.

Pathogenicity.—The spirillum was pathogenic for guinea pigs and rabbits during the first two weeks after its isolation, the animals dying in from twenty-four to forty-eight hours after hypodermic injection of 2 cubic centimeters of a broth culture or of 1 cubic centimeter intraperitoneally. After being isolated for a longer time, the organism lost its pathogenicity.

CONCLUSION.

The cultural characteristics show the spirillum most probably to be a new species, for which I propose the name of *Spirillum zeylanicum*.

ILLUSTRATIONS.

PLATE I. Bronchoöidiosis. Preparation of sputum stained by Leishman's method.
II. Epidermophyton rubrum, sp. nov. Fig. 1. Sabouraud's agar. Fig. 2. Saccharose agar.

PLATE I.

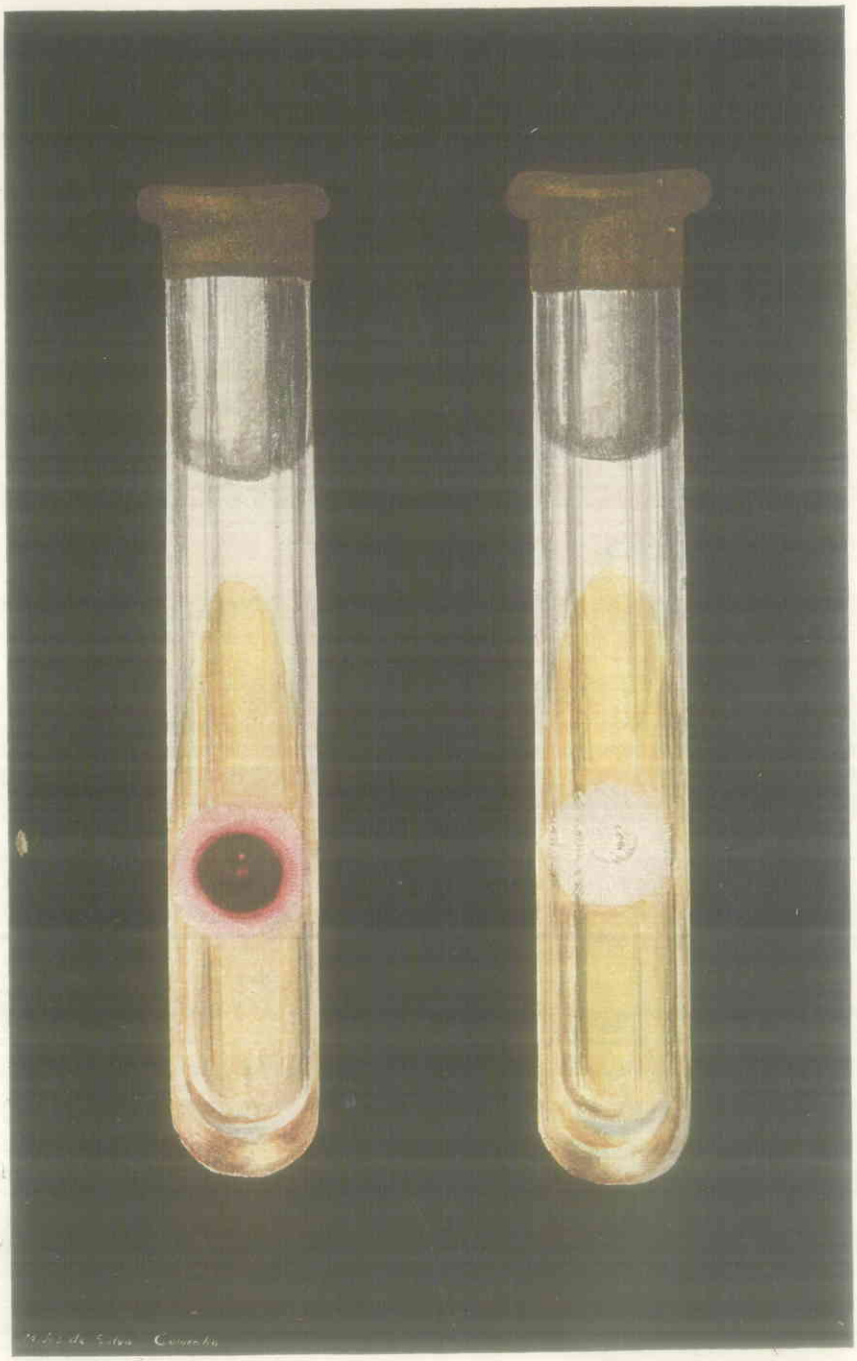

FIG. 1. FIG. 2.

PLATE II.

NOTE ON AN INTESTINAL FLAGELLATE IN MAN.[1]

By Aldo Castellani[2] and Albert J. Chalmers.[3]

Recently we have observed a flagellate in the stools of cases of agchylostomiasis suffering with diarrhœa. A brief description of the organism is as follows:

Fresh preparations.—The parasite is extremely motile in fresh preparations from the liquid stools. Two forms are generally present—a slender, and a larger, more rounded one. It measures about 8 to 15 μ in its greatest diameter, but the shape varies very much because the parasite is capable of amœboid movements, although no true pseudopodia are emitted. Two long flagella originate from one pole by means of which locomotion takes place. There is no evidence of any undulating membrane nor of contractile vacuoles. The protoplasm is homogeneous, but a few vacuoles may be observed in fresh preparations. The nucleus is not visible.

Stained preparations.—In preparations stained by Romanowsky's method the parasites appear globular, nearly round, or pear-shape, and the protoplasm is of a bluish color; a small, rather eccentric, approximately round nucleus is visible, which is rich in chromatin. In some individuals one or more other small chromatic granules may be observed in the protoplasm in addition to the nucleus. One of these chromatic masses often is situated close to that pole of the parasite from which the flagella originate. In successful preparations two flagella are visible which stain a pinkish or purplish color.

Cultures.—The flagellate can be grown in symbiosis with bacteria in various liquid media and in the water of condensation of several solid media.

Acid agar, maltose-agar, serum.—The organism remains alive three or four days in the water of condensation of the tubes inoculated directly from the stools, but it does not multiply and cultivation generally does not succeed in transplantation.

Saccharose agar.—The parasite remains alive for several days, but subcultures fail in the majority of cases.

Sabouraud's agar, acid maltose-agar (2 to 4 per cent), albumen-agar lactose-agar (2 to 4 per cent).—The parasite remains alive and multiplies in the water of condensation for from eight to ten or more days. Subcultures are successful. At present we have the thirty-second subculture and the parasite apparently grows

[1] Read at the first meeting of the Far Eastern Association of Tropical Medicine held at Manila, March 12, 1910.

[2] Professor of Tropical Medicine and Lecturer on Dermatology Ceylon Medical College; delegate from the government of Ceylon.

[3] Registrar and Lecturer on Pathology and Animal Parasitology, Ceylon Medical College.

as vigorously as it did four months ago. We made subcultures of the original strain every three or four days.

Nutrose-agar (2 to 4 per cent).—Probably this is the best medium. The flagellate remains alive for more than two weeks in the water of condensation, and in this medium it appears to be capable of being taken in subculture for an indefinite number of transplants. The tubes should not be capped. We generally make subcultures of the parasite twice a week.

Broth peptone water.—The flagellate dies out within two or three days in the tubes inoculated directly from the stools or from cultures.

Albumin salt solution.—The parasite may be kept alive for a long time in Grassi's albumin salt solution (albumin, 10 cubic centimeters; 5 per cent salt solution, 90 cubic centimeters).

Nutrose broth (2 to 4 per cent).—The germ multiplies and is capable of being transplanted for an indefinite number of times.

CONCLUSION.

In the stools of patients in Ceylon suffering from agchylostomiasis we have observed a flagellate which is pear-shaped or rounded, measuring from 8 to 20 μ in diameter, possessing two flagella, an undulating membrane, and capable of amœboid movements. It is easily cultivated together with bacteria on several media, the best of which is apparently nutrose-agar or nutrose broth. The developmental stages and the methods of reproduction have not as yet been studied and therefore the exact zoölogical position of the parasite can not be defined. We propose to classify it provisionally under the genus *Bodo* and to name it *Bodo asiaticus*.

In two cases in which *Bodo asiaticus* was present another flagellate was also observed. We were not able to cultivate this organism. It is rounded or fusiform, measuring 10 to 15 μ in length, with one flagellum originating at each pole. In fresh preparations it is actively motile. It may present vacuoles, but these are not contractile. In stained preparations a well-marked, though small nucleus which is rich in chromatin, can be distinguished.

ILLUSTRATION.

PLATE I. *Bodo asiaticus* sp. nov.

PLATE I.

SOME CLINICAL ASPECTS OF MYCETOMA, AN UNUSUAL FORM OF CALLOSITY COMPLICATING IT.[1]

By A. Hooton.[2]

The Province of Kathiawar, on the west coast of India, to which the following observations refer, is noted for the prevalence of mycetoma. During the past two years 26 cases have come under my observation. How this incidence contrasts with hospitals in other regions markedly affected by the disease I am not aware, but it is very much in excess of the prevalence in any other station of which I have personal experience. An interesting feature in this connection is the coincidence of actinomycosis in the same area. At the Medical Congress held in Bombay last year I reported two cases of that disease, which, so far as I know, has only been once previously noted in India in man, namely, by Doctor Powell, about ten years ago in Assam. There is so much in common pathologically between mycetoma and actinomycosis that it is not surprising to find them occurring in the same district. Indeed, it is somewhat remarkable that this is not more often the case.

Another unusual condition which impressed me early in my service at the Rajkot Hospital, was a type of multiple callosity which I do not remember to have seen elsewhere. Unlike the ordinary corn, these callosities occur in parts of the sole not especially subject to pressure, and they are also very much more extensive, so that in order to extirpate them it is often necessary to excavate quite a large quantity of tissue. My present observations are based partly on these callosities and their occurrence side by side with mycetoma, and I think that the local prevalence of the latter disease and the occasional conjunction of the two conditions in the same subject afford some ground for the opinion which I have formed that the one is merely a different phase of the other; that the callosity, in fact, is due to a horny degeneration or atrophy of a patch of mycetoma. Additional weight is lent to this view by the fact that the history is frequently the same. The patient often dates both undoubted

[1] Read at the first meeting of the Far Eastern Association of Tropical Medicine, held at Manila March 12, 1910.

[2] Major, I. M. S., Rajkot, Kathiawar, India; delegate from the government of India.

mycetoma and callosities to a prick of a thorn. The photograph (Plate I, fig. 1) shows a case in which there were many callosities on the soles of both feet; Plate I, fig. 2, the two conditions occurring side by side in the same foot; and Plate I, fig. 3, a distinct patch of mycetoma with another internal to it which, I think, is progressing toward a condition of horny degeneration. As regards the disease shown by Plate II, fig. 4, I do not wish to discuss the point; it represents a condition which I have never observed before, and I should be very glad if light could be thrown upon it.

Another stage of the disease, the existence of which has been denied by some authorities, is shown in Plate II, figs. 5 and 6. I am aware that secondary deposits in the lymphatic glands have been previously reported, but these are the only two cases which I have personally seen. In this form mycetoma would appear to be as dangerous as and more rapid in its spread than some varieties of carcinoma; but fortunately such a development is very rare. Recurrence at the site of amputation is apparently even more rare, and I have only once seen a case in point; in this the growth recurred in the flop after a Syme's amputation. My own experience in all cases where there seems to be any reasonable prospect of extirpating the fungus in that way leads me to excise the growth, in preference to sacrificing the foot, even though it may be impossible to allow a very liberal margin of sound tissue. The results of treatment are satisfactory in ordinary cases, and in the malignant type probably even amputation would fail.

Of the 26 patients above referred to, 18 were males, and 13 cultivators by occupation. Fifteen of the growths discharged black granules and 11 yellow granules.

ILLUSTRATIONS.

PLATE I.

FIG. 1. *Showing callosities.*

Male, 38, Hindu, cultivator. Says the corns first began to appear two years ago. He attributes the condition to injury by thorns, which, he says, was succeeded by induration of the skin. Distinct callosities are visible in the photograph, and it will be noticed that they do not all correspond to the areas most subject to pressure, as in the case of ordinary corns.

FIG. 2. *Showing mycetoma with callosities.*

Another specimen is available, which shows this combination equally well, but is not figured. Male, 20, Hindu, cultivator. He states that a year ago, while working in the fields, a thorn entered his right foot, at the base of the second and third toes. An abscess formed, and subsequently a hard, brawny swelling, which later burst and discharged black granules. The photograph shows an undoubted mycetoma, discharging black granules, in the situation indicated above; and several callosities, resembling those of fig. 1, can be seen scattered over the sole of the foot.

FIG. 3. *Showing mycetoma, with an adjacent patch, apparently mycetoma degenerating into a callosity.*

Male, 33, Hindu, corn and general dealer. He states that about a year ago a hard swelling appeared about the middle of the outer border of the right foot, which ultimately broke down and discharged black granules. Shortly before the swelling was noticed he had struck his foot against a stone, and he attributes the disease to that injury. In the photograph an undoubted mycetoma can be seen, which discharged black granules. Internal to this is an indurated patch, which was apparently mycetoma undergoing indurative changes, and which probably (it is suggested), if left alone, would have developed into a callosity like those seen most typically in fig. 1.

PLATE II.

FIG. 4. *Showing a diffuse induration of the sole of the foot, possibly a degenerative phase of mycetoma.*

Male, 20, Hindu, clerk. He states that about five years ago he noticed a small corn (*kapasi*) at the middle of the sole of the right foot. This was followed by others, and gradually the greater part of the sole became covered with a horny growth. No discharge, granular or otherwise, was ever noticed.

On excision, the growth was sent to the Imperial Research Institute, Kasauli, for examination. The report received stated that the microscopic appearances were those of a chronic granuloma, resembling mycetoma, but that no mycelial elements could be discovered.

FIG. 5. *Mycetoma, showing secondary deposits in inguinal glands.*

Female, 40, Hindu, no occupation. She states that about a year ago she first noticed "corns" on the sole of the right foot, which she at first tried to remove. Swelling appeared at the groin shortly after the commencement of the disease in the foot. The photograph shows a well-marked mycetoma, which discharged yellow granules, and there are three sinuses in the groin, in the pus from which similar granules appear.

FIG. 6. *Mycetoma, showing secondary deposits in the inguinal glands.*

Male, 35, Hindu, cultivator. He dates the commencement of the disease from the prick of a thorn in the sole about three years ago, and states that in this and other places "corns" formed. The first swelling in the groin is stated to have appeared three months after the first symptoms. The photograph shows a well-marked mycetoma of the right foot, with a similar growth spreading upwards and downwards from the groin into the abdomen and thigh. Yellow granules are discharged from sinuses in both situations.

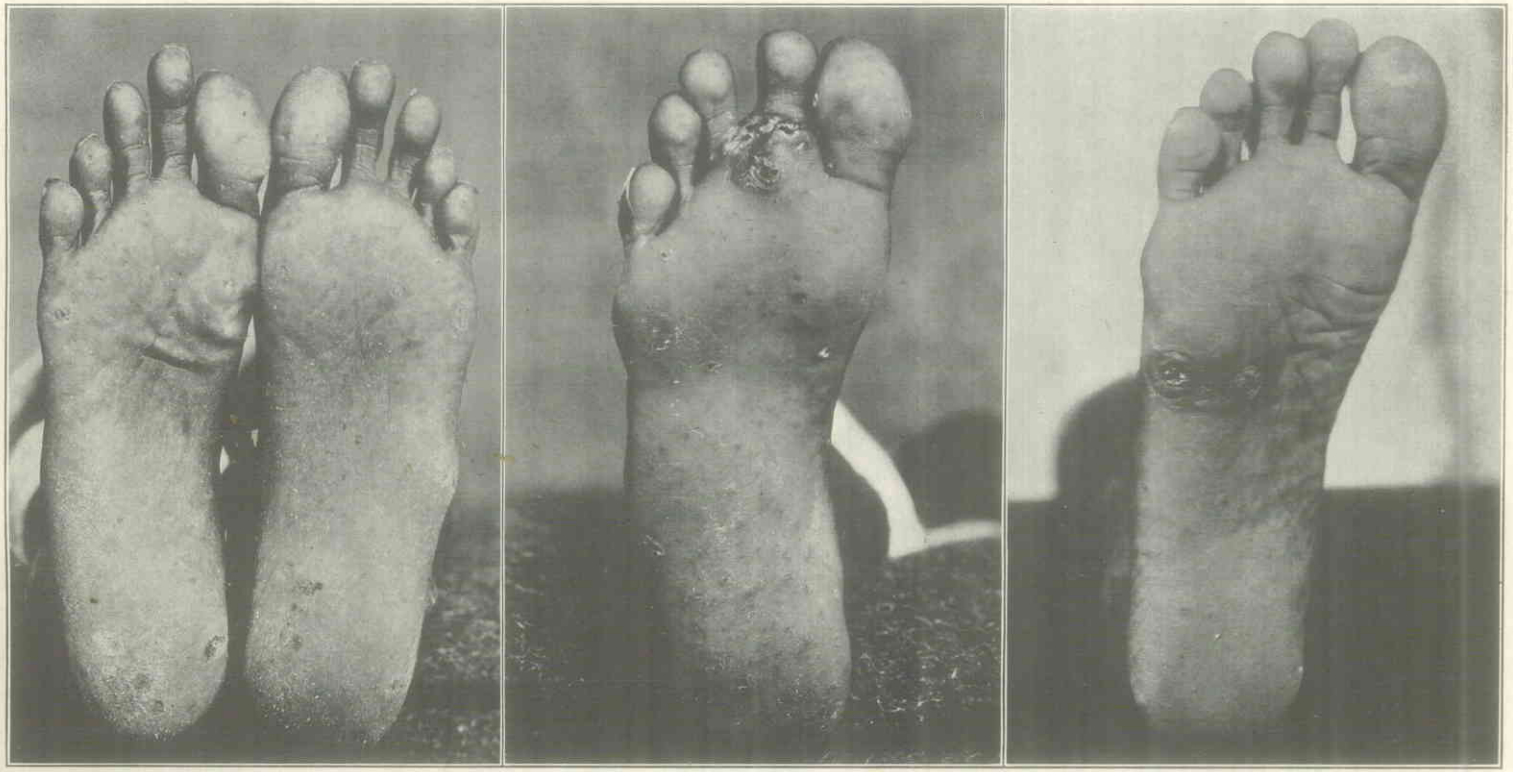

FIG. 1. FIG. 2. FIG. 3.

PLATE I.

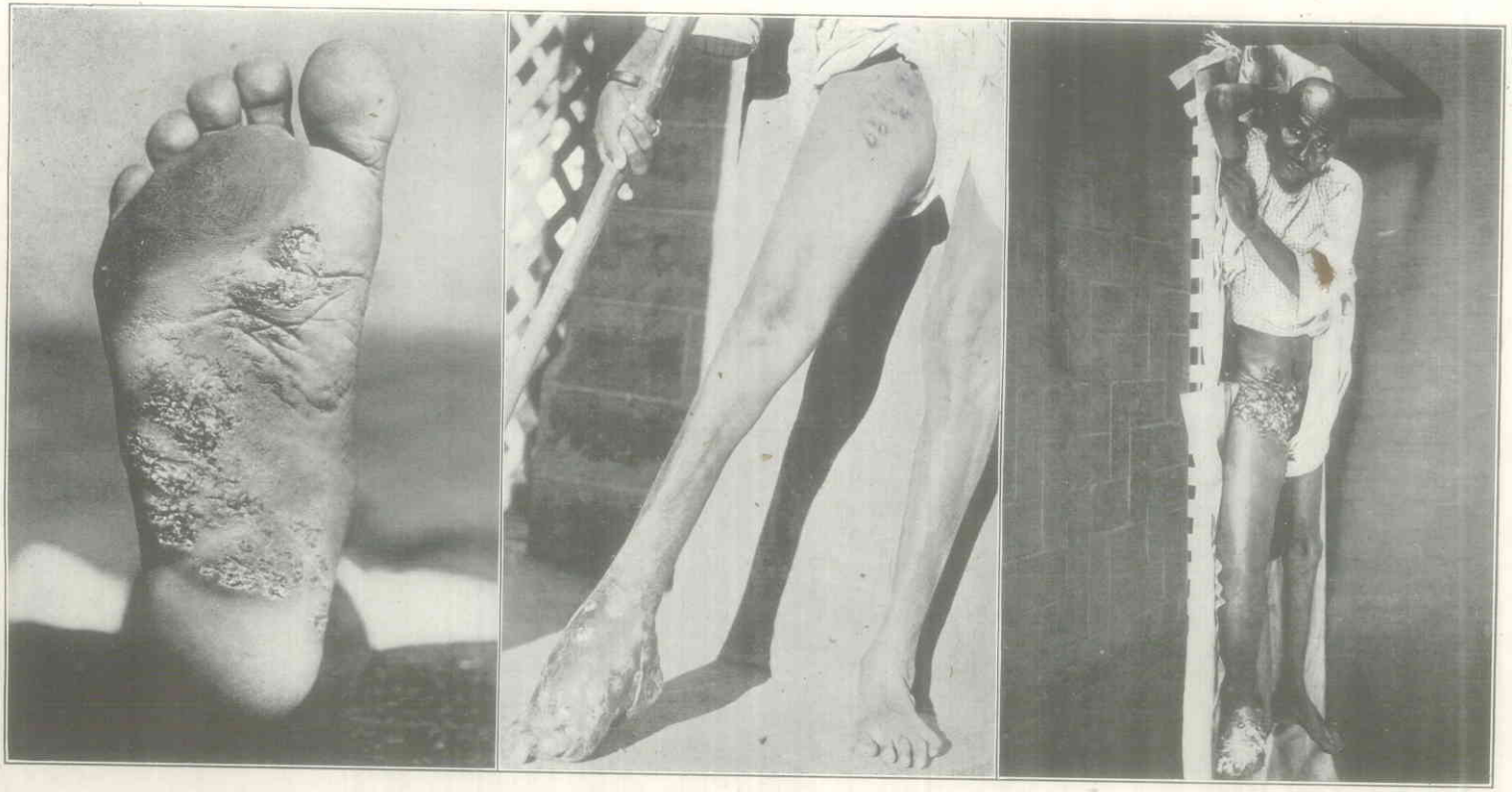

FIG. 4. FIG. 5. FIG. 6.

PLATE II.

THE PREVENTION AND TREATMENT OF AMŒBIC ABSCESS OF THE LIVER.[1]

By LEONARD ROGERS.[2]

During the last few years much progress has been made in our knowledge of the pathology of tropical liver abscess, in which the workers in the Philippine Islands have played a noteworthy part. Nevertheless, I venture to hope that, owing to the abundance of opportunities for investigating this subject in Calcutta, my experience of the last ten years' work there may not be without interest, more especially with regard to the practical points of the prevention and treatment of the disease based on advancing knowledge regarding its etiology.

RELATIONSHIP OF TROPICAL LIVER ABSCESS TO AMŒBIC DYSENTERY.

As recently as 1902, the opener of a debate on dysentery at the British Medical Association[3] maintained that tropical liver abscess was not closely related to dysentery. At the same meeting I brought forward the results of investigations in Calcutta on this point, in a series of cases in which both clinical histories and post-mortem records were available, with the following results:

Relationship of dysentery to tropical liver abscess.

	Total.	Per cent.	
Clinical and post-mortem evidence of dysentery	35	55.5	76.18
No history, but post-mortem evidence of dysentery	13	20.63	
History, but no post-mortem evidence of dysentery	9	14.3	90.48
No history or post-mortem evidence of dysentery	6		9.52

The above cases include a number of old records dating back to 1872, when the relationship of the two diseases was less well known, so dysentery may not always have been sought for and recorded. If we take the more recent post-mortem records of the last ten years, a considerable majority of which have been performed by me, we obtain the following figures.

[1] Received for the first biennial meeting of the Far Eastern Association of Tropical Medicine, held at Manila March 5–14, 1910, after the close of the sessions.

[2] Professor of pathology, Medical College, Calcutta.

[3] *Brit. Med. Journ.* (1902), 2, 841.

Bowel condition in fatal cases of liver abscess.

	Total.	Per cent.
Amœbic dysentery present	35	77.8
Scars of old dysentery present	9	20.0
No evidence of former dysentery	1	2.2

Evidence of dysentery, always of the amœbic type, is thus seen to be almost constantly found after death from amœbic abscess of the liver, while the few exceptions can readily be explained on the assumption that a mild latent infection had completely healed before death from the hepatic complication, leaving no very evident scarring behind.

Once more, an analysis of the excellent clinical notes on liver obscess treated in the European General Hospital at Calcutta during the last nine years has furnished the following data:

		Total.		Per cent.	
Dysentery	In hospital	18	36	72	86
	Within 3 months	10			
	Over 3 months ago	8			
Diarrhœa only			7	14	
No diarrhœa			7	14	

There was thus a history of dysentery or diarrhœa in 86 per cent of the cases of liver abscess, while we have already seen that these symptoms may be absent even when amœbic ulceration of the large bowel, commonly limited to the cæcum and ascending colon, are actually found post-mortem.

A further study of the cases in the Medical College Hospital demonstrated that in at least 80 per cent the symptoms of dysentery had preceded the formation of liver abscess, while when the bowel symptoms supervened after signs of acute hepatitis, it is doubtless only a recrudescence of old intestinal trouble.

For these various reasons I hold that amœbic dysentery, either active or latent, invariably precedes amœbic hepatitis, which is secondary to it by infection through the portal system. In 1903 [4] I demonstrated that the earliest minute amœbic multiple abscesses of the liver commence in the terminal branches of the portal veins, and I have several times since found similar small multiple abscesses of the liver containing amœbæ, but no bacteria or cocci.

THE PRESUPPURATIVE STAGE OF AMŒBIC HEPATITIS.

The vast majority of patients coming to hospital with an abscess of the liver give a history of fever, with or without pain in the hepatic region, lasting for several weeks and not rarely for several months. They have usually been given quinine and other drugs without avail, the true nature of the disease only becoming apparent with an increased prominence of the localized symptoms. When examining the blood of a large

[4] *Ibid.* (1903), 1, 706.

number of consecutive fever cases in the Calcutta European Hospital, I realized that the fever of the presuppurative stage of amœbic hepatitis could almost invariably be recognized by the occurrence of a leucocytosis, usually with no marked increase in the proportion of the polynuclears. I had previously come to the conclusion that active or latent amœbic ulceration of the large bowel always preceded liver complication, and that ipecacuanha is of great value in amœbic dysentery, a point in which Indian experience is at variance with the general opinion in the Philippine Islands, although Simon in New York and Dock in New Orleans are now converts to the view which Sir Patrick Manson and the writer have long advocated. It was but a simple step forward to try the effect of full doses of ipecacuanha in the early stages of amœbic hepatitis, with a view to curing the exciting cause, namely, the ulcers in the large intestine, as had indeed been done empirically many years before by Maclean and Norman Chevers, although with the increasing vogue of the saline treatment in dysentery the use of ipecacuanha had fallen largely out of favor even in the latter disease and had been almost entirely neglected in hepatitis of recent years. I have already published several papers[5] illustrating the marvellous effects of this drug in preventing acute hepatitis passing on to liver abscess, sanctioned even when experienced surgeons were convinced that suppuration had already taken place. A consecutive series will be found in my work on Fevers in the Tropics, while I now have notes of several scores of equally striking cases, which it is unnecessary to relate. It will suffice here to say that during the last four years no patient has developed an amœbic liver abscess under treatment for hepatitis in the 100 beds for males in the Calcutta open wards of the Calcutta European Hospital, although this was formerly a frequent occurrence, while the number of patients admitted with an abscess of the liver has also fallen considerably. Even more striking evidence of the value of this plan is furnished by the returns of the British army in India, for during the two years which followed the publication of my first series of cases the mortality from liver abscess in English troops in India has fallen by 60 per cent, although stationary for the previous thirteen years. With the wider adoption of this treatment in the earliest stages of tropical hepatitis, I feel sure even better results will be obtained in the army, as the patients come under skilled medical observation at the beginning of their illnesses. It is already abundantly clear that I did not exaggerate when I wrote that amœbic or tropical liver abscess is an easily preventable disease in the great majority of instances, and the occurrence of amœbic suppuration in the liver should cause serious questions in the mind of the medical man in whose hands it has been allowed to develop.

[5] *The Therap. Gaz.* (1909).

Diagnosis.—Now that I have shown that leucocytosis of a marked degree occurs in the readily curable presuppurative stage of amœbic hepatitis, it is clear that we can obtain no aid from an increase of the leucocytes as a sign of the actual formation of an abscess in the liver in a doubtful case. Complete fixation of the diaphragm and rarely an increased density of the liver shadow, as seen with the X rays, may be present in cases which clear up under ipecacuanha. I have also seen a local swelling in the epigastrium and even œdema over the ribs in cases rapidly cured in this way. In fact, I know of no certain symptom of the formation of pus within the liver except a fluctuating swelling in the hepatic region. It is for this reason that exploratory puncture is so often performed in acute hepatitis for the confirmation of suspected liver abscess, only too often with a negative result; nor is this operation the harmless procedure it is frequently represented to be. I know of several fatalities from hæmorrhage into the abdominal cavity resulting from it, while Lieutenant-Colonel Hatch, I. M. S.,[c] has done a public service by having had the courage to publish a series of six such cases in his experience, occurring in the Bombay Presidency. Moreover, this disaster is most likely to occur in cases in which no abscess is found, in some of which it was proved post-mortem that no pus had formed in the liver. It is especially in the acutely congestive presuppurative stage that serious bleeding follows exploratory puncture of the liver; yet it is often most important to detect and deal with an abscess in the liver before it has clearly revealed itself by its large size or through implicating surrounding organs, and hitherto the needle was the only means of deciding if suppuration had taken place or not. Fortunately, we are now in possession of a much simpler and safer plan, for if the disease is still in the presuppurative stage the fever, pain, and liver enlargement all rapidly yield to ipecacuanha, which is as much a specific for amœbic hepatitis in the early stage as quinine is for malaria. No harm results from a few days' delay if pus has already formed, while the danger of further abscess formation will be greatly reduced by the drug treatment. If, however, fever continues for a week or more without material diminution, and especially if the local pain remains, an abscess has probably already formed and exploratory puncture may now be performed with much less risk, as the general congestion of the liver will have been greatly lessened by the ipecacuanha. Since this rule has been followed in the Calcutta European Hospital negative exploratory punctures for liver abscess have become as rare as formerly they were frequent. In the Medical College Hospital explorations are still performed by the surgeons in doubtful cases, without a previous trial of ipecacuanha, and negative

[c] *Indian Med. Gaz.* (1898).

results are still obtained in cases which subsequently recover completely on ipecacuanha.

THE STERILITY AS REGARDS BACTERIA OF AMŒBIC ABSCESSES OF THE LIVER.

The most important point in regard to treatment is the absence of bacteria from the pus of amœbic abscesses of the liver in the great majority of cases. In the first series in which I examined the pus obtained at the time of the operation, I found two-thirds to be sterile, but this was an underestimate, as the aspiration bottles were not always free from bacteria. In a recent series, in which the pus was received direct from the aspirating canula into a sterile test tube, no less than 75 out of 87 consecutive cases, or 86 per cent, were free from bacteria both microscopically and on culture. However, in a few cases very numerous cocci and bacteria are found, occasionally including streptococci. Such cases have a worse prognosis than abscesses containing only the amœba. The great majority, however, of tropical liver abscesses are solely due to the protozoäl organism.

THE FREQUENCY OF BACTERIAL INFECTION AFTER THE OPEN OPERATION FOR PRIMARILY STERILE AMŒBIC ABSCESSES.

When examining pus from opened liver abscesses for amœba, I observed that bacteria and cocci were almost invariably present. I therefore made cultures in a series of cases from the pus obtained in sterile test tubes at the time of opening and again a few days later. In a large number of observations made during the last two years at the Medical College Hospital at Calcutta in no single case did a primarily sterile abscess remain free from infection for as long as three days after being opened and drained in the ordinary way; nor is this surprising when we remember that the sterile, blood-serum-like contents form an ideal culture medium for the organisms, which must inevitably enter from the air at the operation and on subsequent dressings, apart from the frequency with which the copious discharges soak through the coverings, and thus allow of contamination. The organisms will commonly be of but slight virulence, but suffice greatly to prolong the period of exhausting discharges and retard the healing of the wound. In support of this contention it may be well to quote the following remarks of Maj. G. C. Spencer, R. A. M. C., professor of military surgery, Royal Army Medical College.[1] Regarding the high mortality of the open operation he writes:

> The chief cause of this high mortality, apart from the presence of more than one abscess, or extreme debility of the patient before operation, is undoubtedly infection of the abscess cavity by pyogenic organisms through the open wound. This is extremely difficult to prevent, no matter how much care is taken. . . .

[1] *Journ. Roy. Army Med. Corps* (1909).

The great majority of amœbic abscesses are sterile when first opened, and every surgeon with Indian experience is familiar with the usual course of fatal cases. The patient does well for the first few days after operation, then infection occurs, the temperature goes up again, and death from septic poisoning slowly but surely follows.

THE TREATMENT OF A STERILE AMŒBIC ABSCESS OF THE LIVER BY REPEATED ASPIRATIONS AND INJECTIONS OF QUININE INTO THE CAVITY WITHOUT DRAINAGE.

In abscesses due to the ordinary pyogenic bacteria there is no difference of opinion as to the necessity of early opening and drainage. A similar line of treatment in large, cold tubercular abscesses, however, has sometimes been followed by a disastrous secondary infection. Tropical abscesses of the liver for many years past have been almost universally treated by free incision and open drainage, exactly as in ordinary septic abscesses due to bacteria. The results can not be said to be brilliant, for in the Calcutta hospitals the mortality among several hundred cases, treated by very experienced surgeons during the past thirteen years, has been 60 per cent. Now that we know that this form of abscess is caused by a protozoäl organism, and the vast majority of them are free from bacteria when first opened, although infection almost invariably follows their free incision, it is worth considering if some simpler and safer method of treating the disease can not be found. On ascertaining that tropical liver abscesses always contain amœbæ and are usually primarily sterile, I set to work to test the effect of drugs on the causative protozoa in the walls of liver abscesses, post-mortem, and in 1902 recorded the fact that a 1 to 500 solution of quinine would readily destroy the amœbæ under these conditions. I therefore suggested the treatment of bacterially sterile liver abscesses by withdrawal of as much pus as possible through an aspirating needle and the injection of 1.30 grams (2 grains) of quinine in solution into the cavity, no incision or drainage being used. In 1906 I reported with Capt. R. P. Wilson, I. M. S.,[8] two cases successfully treated by this plan, since which several surgeons have recorded similar results. Major Spencer early in 1909 published several, in one of which three aspirations and injections were necessary, while in the other two a single operation sufficed, and he advised the adoption of my plan in all cases, as even if it fails no harm is done, and the patient may be in a better condition to stand the more serious open operation.

During the past year Maj. F. O'Kinealy and C. R. Stevens have kindly given my method a prolonged trial at the Calcutta Medical College Hospital with most encouraging results. The latter surgeon will shortly publish his own conclusions, so I will only here tabulate the results of the cases I have been able to watch in the several Calcutta hospitals during the last few years.

[8] Brit. Med. Journ. (1906) 1, 1397.

Summary of cases treated by aspiration and quinine injection only.

		Abscess cured.	Abscess fatal.
Abscess not opened	Cured	16	
	Abscess cured, died later of dysentery	1	
	Abscess cured, died later of pneumonia	1	
	Died of liver abscess		3
Abscess opened later	Cured	3	
	Died		3
Total		21	6
Abscess evacuated through the thoracic wall			25
Abscess evacuated through the abdominal wall			2

The site of the evacuation of the pus is important, because I find that the mortality of cases treated by the open operation is but 40 per cent when the incision is through the abdominal wall, but no less than 73 per cent when it is through the thoracic wall. The death rate was but 12 per cent in abscesses of the left lobe opened through the epigastrium, owing to their being readily recognized and dealt with while still small. In the above series the death rate among 25 cases evacuated through the chest wall was 24 per cent, or just one-third of the mortality of similar cases treated by the open operation. The three cases in which a fatal result occurred after aspiration and quinine injection alone all had large abscesses, the patients being nearly moribund, and the open operation would almost inevitably have been rapidly fatal. On the other hand, several abscesses containing from 1.5 to 3 liters (3 to 6 pints) of pus were successfully dealt with by my plan, including some in which it was considered that the open operation would have given little or no chance of recovery, one containing 3.3 liters (112 ounces) of pus. In one case the patient died of dysentery three and one-half months after cure of the liver abscess, my suggestion to give ipecacuanha at the time the abscess was first aspirated not having been adopted. In another case no less than 2.5 liters (86 ounces) of pus were aspirated at the first operation, nine days later 532 cubic centimeters (18 ounces) were obtained and seven days later still only 300 cubic centimeters (10 ounces) were evacuated, quinine being injected at each aspiration. After another eight days only 150 cubic centimeters (5 ounces) of thin bile, without pus, were withdrawn, so no quinine was injected. A few days later the patient died unexpectedly and at post-mortem, left apical pneumococcal consolidation of the lung was found, quite independently of the hepatic trouble. The liver abscess was found to have contracted so as to hold only 75 cubic centimeters (2½ ounces) of thin bile with no pus or amœbæ, the contents being sterile, as they had been throughout. The fibrous wall was smooth and nearly half an inch in thickness, encystment having taken place. The patient had been given a course of ipecacuanha, and the cæcum

showed scars of recently healed ulcers, together with a few depressed slits of those almost healed. Another remarkable case was one of Major O'Kinealy's, in which no less than 3 liters (6 pints) of pus were aspirated from the liver of an Indian patient. This pus was found to be sterile as regards bacteria. Five days later 1,064 cubic centimeters (36 ounces) were withdrawn, and 2.66 grams (40 grains) of the soluble bihydrochloride of quinine injected. The patient improved steadily and put on 8½ kilograms (18½ pounds) weight in five weeks, recovering completely. Such cases speak for themselves.

In carrying out this method, the skin at the seat of puncture must be thoroughly sterilized to prevent bacteria being carried into the abscess cavity. The T tube of the exhaust bottle should have a large caliber to allow thick pus to pass, the cavity being emptied as far as possible, and some of the pus put in a sterile test tube for bacteriological examination. A previously boiled solution of the bihydrocholoride of quinine, 2 grams to 100 cubic centimeters (10 grains to the ounce) is next injected into the cavity through the canula, which is then withdrawn and collodion applied externally. If only a few cubic centimeters (ounces) of pus have been found, 60 cubic centimeters (2 ounces) of the quinine solution will suffice, but if a pint or more of pus has been removed, 120 cubic centimeters (4 ounces) should be injected. In some cases the temperature at once falls and all the symptoms rapidly subside, but more frequently the process has to be repeated after a week, while third and fourth aspirations are sometimes required in large abscesses. If a previously present leucocytosis completely subsides, little or no pus is usually obtained at a second operation, but the continued presence of leucocytosis is an indication for further aspirations, quinine being injected each time. In Major Spencer's cases the hydrobromide of quinine was used successfully. In the rare cases in which the aspirated pus is found to be swarming with bacteria the abscess must be opened. Further experience is required to lay down the exact indications for this method, but the success already obtained is sufficient to make it advisable to give the patient the benefit of a trial of this simple and safe mode of treatment in all cases in which there is no definite contraindication before resorting to the much more serious open operation.

STERILE SYPHON DRAINAGE OF LIVER ABSCESS.

The practical impossibility of maintaining sterility in the Tropics after the open operation for liver abscess, taken with the occasional failure of my plan of aspiration and quinine injecton, suggested to me the advisability of devising a method of sterile drainage, combined with quinine irrigations. For this purpose I got Messrs. Down Brothers, of London, to make for me the flexible sheathed trocar.[9] It is made in various sizes and can be used as an aspiration trocar, the abscess being first located with the ordinary small-sized trocar if its position is not

[9] *Ibid.* (1908), 2, 1246.

accurately known. After the cavity is evacuated a piece of tubing of large bore is connected with the end of the flexible sheath, and carried into a bottle of antiseptic lotion under the bed, siphon drainage being thus established. The sheath is so flexible that it can safely be left in the cavity and used as a drainage tube. By means of a Y-shaped silver tube connected with pressure tubing the aspirator can be applied daily to the flexible sheath, and any thick pus which is not draining can thus be withdrawn. Through the other limb of the Y tube, with a sterile glass syringe, sterile quinine solution is injected daily, to kill the amœbæ in the wall of the abscess. The discharge rapidly lessens and the cavity contracts. Thus, in the first case in which this method was used in Calcutta by Capt. J. G. Murray, I. M. S.,[10] in less than a week a cavity originally containing about 500 cubic centimeters (1 pint) of pus would admit only 15 grams (one-half ounce) of the quinine solution. After two weeks (an unnecessarily long time, to be on the safe side) the canula was withdrawn, leaving a 10 centimeters (4-inch) sinus. In three days the sinus had healed up to the surface with only a few drops of serous discharge, and in a week from the removal of the canula the skin had healed over and the patient left the hospital, striking contrast to the slow process attending infected liver abscess wounds after the open operation. The patient was discharged from the hospital in less than half the time that any similar liver abscess evacuated through the chest wall had been cured by the open operation in the European Hospital during the last nine years. The great advantages of sterile siphon drainage combined with sterile daily quinine irrigations is thus clearly established, but the marked success of repeated aspirations and quinine injections recently obtained promises greatly to limit the necessity for the employment of my flexible sheathed trocar.

THE USE OF IPECACUANHA IN THE AFTER-TREATMENT OF AMŒBIC LIVER ABSCESS.

Lastly, I would urge that every patient operated on for amœbic liver abscess should be given a course of full doses of ipecacuanha as soon as possible, with the view to healing the ulcers in the large bowel, which have originated the hepatic trouble and are often latent and give rise to no symptoms. This will greatly lessen, or entirely prevent, the formation of further liver abscesses, the occurrence of which, during the convalescence after operation for a collection of pus, is one of the most trying complications the surgeon in the Tropics is liable to meet with. If the operation is resorted to, the cavity should also be washed out with sterile quinine lotion daily, as this will rapidly lessen the discharge if no serious bacterial infection has resulted. As an example of the value of this meausure, I may mention the case of an European who had been

[10] *Loc. cit.*, 1330.

submitted to four operations for liver abscesses in as many months and was still suffering from severe pain and high fever, being in a very precarious condition. He was at length put on ipecacuanha, and quinine irrigation was adopted. The next day his pain had almost gone, the temperature rapidly fell, and he began to recover from that time, although several weeks were required for the healing of his extensive wounds. I have seen him in very good health a year after leaving the hospital, and he has recently passed a medical examination for employment as an engineer on a large railway. He was so impressed with the immediate relief afforded to him by the ipecacuanha that he continued to take it daily for a year after his recovery. In fact, I attribute not a little of the recent improvement in the results of the treatment of liver abscess in the Calcutta hospitals to the adoption of a routine course of ipecacuanha in the after-treatment of liver abscesses, however they may be dealt with.

INTESTINAL AMŒBIASIS WITHOUT DIARRHŒA.

A STUDY OF FIFTY FATAL CASES.

By W. E. MUSGRAVE.

(From the Biological Laboratory, Bureau of Science, Manila, P. I., and the Department of Clinical Medicine, Philippine Medical School.)

Notwithstanding the fact that such careful and experienced observers as Osler, Dock, Councilman, Laffleur, Kartulis, Tuttle, and many others have mentioned the absence of diarrhœa in certain severe and even fatal cases of amœbic infection of the colon, the fact does not appear to have received the general recognition which its importance deserves.

In 1904 Musgrave and Clegg again called attention to this condition, having in several articles which have appeared since that time, reported cases and emphasized its occurrence. The purpose of the present paper is not to establish a new fact, but to show that the prevalence of amœbic infection of the colon without diarrhœa is of sufficiently frequent occurrence to deserve careful consideration by clinicians and to make evident the necessity of altering our conception of the disease to conform with the acceptance of such observations.

In selecting the 50 cases for this report, only those in which the clinical observations were of sufficient accuracy for publication and in which the diagnosis was confirmed by autopsy have been used. Of the 50 cases, 8 were foreigners and 42 natives of the Philippine Islands, 47 were males and 3 females. The causes of death were as follows:

Three from peritonitis following perforation of the appendix—two of these produced by amœbic ulceration, the other by an unknown cause, not amœbic.

Four from liver abscesses—one perforating into the right pleura, one into the abdominal cavity, and two were without perforation.

One from acute pericarditis.

Eight from pulmonary tuberculosis, and in three of these abdominal tuberculosis was also present.

Two from chronic æstivo-autumnal fever.

Five from perforation of amœbic ulcers in the large intestine—four times in the cæcum and ascending colon and once in the transverse colon.

Seven from acute beriberi.

Twenty from lobar pneumonia.

Many more could be added to this group of cases in which diarrhœa developed only a few days before death, and then this symptom was often due to intercurrent disease, such as cholera, which caused the exitus. Still another group which might well be classed here includes those patients in which diarrhœa or clinical dysentery developed a few days before death and in which, at autopsy, *advanced* amœbic lesions were found. However, as these cases did show some symptoms of diarrhœa, they are not here considered. In the 50 instances which form the subject of this paper, looseness of the bowels, except from cathartic medicine, was not at any time a symptom. Indeed, in several constipation was a noticeable and constant feature of the disease up to the time of death.

Pathology.—Characteristic amœbic lesions were present at autopsy in all of the 50 cases. These lesions varied in type from those which were just beginning to those showing ulcers having the characteristic extensive destruction of the mucous membrane of the bowel so often seen in cases of amœbiasis of long standing. The lesions were distributed as follows:

Not recorded, 5 cases; confined to cæcum and ascending colon (including 3 of the appendix), 27; entire large bowel (except sigmoid and rectum), 9; rectum, 0; descending colon and sigmoid flexure (alone), 2; transverse colon (including splenic flexure), 2; cæcum, hepatic and splenic flexures, 5.

Other parasites, such as monads, trichuris, hookworms and ascaris were present in several of these patients; the lesions of other diseases have been mentioned above. The duration of the infection, judging from the autopsy findings, varied, but in most instances the lesions indicated processes of long standing.

Symptoms.—The occurrence of general symptoms in these patients varied considerably, and if we except those due to the intercurrent disease were *entirely absent* in some of them. In others there were present one or more clinical manifestations which I have described elsewhere as occurring in latent and masked types of amœbic dysentery. While none of these symptoms may be said to be pathognomonic of amœbic infection, yet when several are present in the same patient, in the absence of any other satisfactory cause, they are strongly suggestive of amœbic infection, especially in zones where this disease is endemic.

Abdominal "aching," usually more or less general, worse at night and early in the morning, and often accompanied by flatulence and occasionally by constipation, is one of the most frequent of the symptoms, but unfortunately this is extremely common among a large class of patients with mild forms of indigestion who are not suffering from amœbic infection. Distension of the abdomen and the discomforts of flatulence are of frequent occurrence. Constipation is a particularly common complaint. In this class of patients the lack of result from ordinary

doses of the usual cathartics may be brought to the attention of the physician, or in other instances the action of these drugs may be unusually severe and prolonged. Loss of weight occasionally becomes a noticeable symptom, but in many instances the nutrition remains good and the patients may even increase in weight. Interference with the appetite is usually first shown by lack of desire for breakfast and this may be accompanied by morning nausea and the accumulation of considerable mucus in the mouth and throat during the night. Active indigestion or dyspepsia are not very common symptoms, but do occur in a certain percentage of the cases.

Excessive perspiration, particularly of the palmar and plantar surfaces, is very frequent, and in many instances the physician is first consulted because of this complaint. The whole chain of symptoms of so-called "Philippinitis" or tropical neurasthenia, characterized by dullness, headache, loss of memory, weakness, desire for sleep, etc., is a rather common condition encountered in these infections, but it is also particularly prevalent in the absence of such parasitic invasion.

Diagnosis.—When we come to study the clinical phenomena shown by this class of cases, it is seen that there is nothing specific or definite in any one, or in all the findings, except the one of the presence of amœbæ in the stools. The patients are of the class sometimes reported as "healthy people with amœbæ in the stools," and, as alluded to in one of my previous papers, it is erroneous to report all of such cases as being healthy or as those "suffering from diseases other than dysentery."

This brings us to the important point which of itself is sufficient excuse for this paper, namely, How are we to diagnose amœbic infection of the bowel during life? Ten years' continuous experience with this disease clinically, in the laboratory and at autopsy, has convinced me that its diagnosis is not possible except through information secured by a microscopic examination of the fæces. Looseness of the bowels in the form of dysentery or diarrhœa has long been the strong diagnostic point, but the facts show it also to be a very unreliable one.

The sygmoidoscope gives valuable and positive evidence of infection in patients with ulceration in the lower part of the bowel, but does not furnish aid in the large percentage of early infections in which the lesions are above the range of this instrument. As a result of careful application of all known diagnostic methods in the infection we have but one constant finding, and that is the presence of amœbæ in the bowel discharge. The question whether the presence of amœbæ in the stools of patients should be considered sufficient evidence of infection for the institution of treatment is still a disputed one. A number of authors agree with the late Professor Schaudinn that there are two easily differentiated species of amœbæ encountered in stools, one a pathogenic parasite and the other a harmless commensal.

I have already discussed this subject fully in other publications.

A QUICK, SIMPLE, AND ACCURATE METHOD OF MAKING DIFFERENTIAL BLOOD COUNTS IN WET PREPARATIONS AND ITS ADVANTAGES IN THE DIAGNOSIS OF SURGICAL AND TROPICAL DISEASES.[1]

By E. R. Stitt.[2]

(From the United States Naval Hospital, Cañacao, P. I.)

In the diagnosis of acute abdominal conditions and almost to an equal extent in other surgical affections, the leucocyte count has the confidence of the surgeon. The same would obtain for the polymorphonuclear percentage were it not for the errors incident to the usual method of making differential counts. A smear equally distributed, a satisfactory stain and good technique are necessary for results that will give true findings. These three factors do not always go together, as any experienced laboratory worker will admit, and for the average man they rarely obtain. Again, almost any variation in the leucocyte percentages can be obtained in the usual easily prepared smear made with some form of spreader on a slide. To make this evident it is only necessary to refer to the usual method of ploughing out the polymorphonuclears to the margins, a method used in preparing smears for determining the opsonic index. I was told by one of our leading American laboratory workers that it was necessary for him to discard several thousand smears of yellow-fever blood, made for the purpose of studying leucocytic percentages in the disease, for the reason just stated.

It has been my experience that the only method of making films which gives fairly accurate findings is that of Ehrlich—the sliding apart of two cover glasses between which the drop of blood has distributed itself in a thin layer. Even with this procedure we meet sources of error such as the difficulty of distinguishing polymorphonuclears from transitionals in those portions of the smear which fail to show a single layer of red cells and in particular by reason of the large number of disrupted cells which are peculiarly common to pathologic blood and

[1] Read at the first biennial meeting of the Far Eastern Association of Tropical Medicine, held at Manila March 12, 1910.

[2] Surgeon, United States Navy.

which show themselves in the form of more or less uniformly stained blotches, the former leucocytic character of which can not be determined. By checking the percentages obtained by the method I shall describe and those secured by the usual means, I have found that in septic conditions these disrupted cells are largely polymorphonuclear, while in such conditions as malaria and dengue they are chiefly transitionals.

The surgeon of the present day always demands a leucocyte count, and the medical man in the Tropics finds it almost equally necessary in differentiating diseases which show a leucocytosis from those which present a leucopenia. Consequently, a distinct saving of time would result if it were possible to obtain other findings in the same preparation used for ascertaining the number of leucocytes per cubic millimeter.

By employing the ordinary technique for making a count of the white blood cells, with the exception that I use a diluting fluid made by adding five drops of Giemsa's stain to 5 cubic centimeters of 2 per cent formalin, I also am able quickly and, I am convinced, accurately to make a polymorphonuclear percentage count, or a complete differential count in addition to that of the leucocytes.

Another advantage is that blood parasites are also perfectly stained, are shown distinctly, and by reason of the larger amount of blood visible in each field, the finding of them is far less tedious than where a stained, dry film is used.

In preparing the Giemsa stain I use the original method by dissolving 0.08 gram Azur II and 0.3 gram Azur II eosin in 25 cubic centimeters of glycerine at 60°C., then adding 25 cubic centimeters of methyl alcohol, allowing the whole to stand overnight and then filtering.

The ordinary commercial formalin and distilled water are used in preparing the 2 per cent formalin solution.

Better results are obtained when the Giemsa solution is added to the formalin just prior to using. The staining power of the mixed formalin and Giemsa begins to diminish after a few hours, therefore it is better to drop the Giemsa solution from a dropping bottle into the formalin in a watch glass at about the time the blood count is to be made. The best results are secured when the mixing in the pipette bulb is done immediately after taking up the blood and diluent.

The usual technique in making the hæmocytometer preparation is employed, a Türck ruling being used. I count the leucocytes in the 3 upper or lower square millimeters, divide by 3 to obtain an average per square millimeter, multiply by 10 for the content of a cubic millimeter and then by 20 for the dilution. (Blood to 0.5, diluent to 11.) This can be done mentally and requires no calculation on paper. Having counted the leucocytes I again go over the same portion of the ruled surface and determine the polymorphonuclears and estimate the percentage of these to the total leucocytes.

It is unnecessary in such counts to have an assistant record the results. Of course, in making a complete differential count it is preferable to have some one tabulate them, or laboriously to do this personally.

The red cells are practically diaphanous and not disintegrated as they are when acetic acid is used as a diluent; consequently it is easy to distinguish the particulars concerning the size, etc., of a particular red cell containing a malarial parasite. Whether it is possible to determine the species of malarial parasite in such a preparation I am unable to state, as I have had only benign tertian and æstivo-autumnal blood to work with since using this method. At any rate I always make an ordinary Ehrlich smear at the same time I take the blood for the white count, so that I have material for further study with a one-twelfth objective should such further study seem to be advantageous.

My best results have been obtained with a one-sixth objective. Higher powers are of course impracticable by reason of the thickness of the cover glass of the hæmocytometer.

The following are the appearances of the various leucocytes.

Eosinophiles.—In these the bilobed nucleus stains rather faintly and the color is greenish-blue. The eosinophile granules show easily as coarse, brick-dust colored particles.

Polymorphonuclears.—The nucleus stains a deep rich violet-blue, but of a less intense color than of the small lymphocyte. The shape of the nucleus is typically 3 or 4 lobed, but even when of the horseshoe shape of a transitional nucleus, it is easily recognizable by the intensity of the nuclear staining. The distinctness of the cell outlines produced by the fine yellowish granulations in the cytoplasm makes the polymorphonuclears very easy of differentiation.

Small lymphocytes.—The nucleus is perfectly round and stains to a deep rich blue. It is almost impossible to make out any cytoplasmic fringe.

Large lymphocytes.—The nucleus here is round and of a lighter blue than that of the small lymphocyte. The cytoplasm is nongranular and sharply defined from the nucleus.

Large mononuclears.—These show a washed-out, slate-colored nucleus which blends with the gray slate-blue staining of the cytoplasm, so that there is an indefiniteness of outline in the more or less irregularly contoured nucleus.

Transitionals.—These have the same characteristics as the large mononuclears, but with a more faintly stained and more indented nucleus. The large mononuclears and transitionals stand out as slate-colored cells without any sharp nuclear definition. When very much degenerated these cells have a greenish hue.

The young ring forms of malaria show as violet-blue areas in the red cell. When half grown or approaching the merocytic stage the containing red cell takes on a faint pink color, thereby differentiating it from the noninfected red cells. At the same time, the parasite is extruded and has the appearance of a violet-blue body projecting from the margin of the red cell. It is as if a blue body were budding from a pink one. The malarial crescents are brought out with the greatest distinctness.

Trypanosoma lewisi in the blood of rats stains quite distinctly. With the comparatively low powers which it is necessary to use, I have been unable to assure myself of chromatic staining.

DISCUSSION ON THE PAPER, "STUDIES ON INFANT MORTALITY," BY DOCTORS McLAUGHLIN AND ANDREWS.

Dr. H. M. Neeb, medical officer of the first class, delegate from Her Majesty's Government of the Netherlands Indies.—Doctors McLaughlin and Andrews have told us in their interesting paper that convulsions bring about the deaths of many children. I wish to inquire if blood examinations were made with the view of determining whether malarial infection was present. I ask this because in our experience in the Netherlands Indies it not infrequently is true that such an infection in children really is the cause of convulsions and of death.

Dr. W. E. Musgrave, of the Biological Laboratory, Bureau of Science, professor of clinical medicine, Philippine Medical School, Manila, P. I.—Doctors McLaughlin and Andrews are to be congratulated on their contribution to the study of the subject of infant mortality. That part of the paper referring to "infantile beriberi" is particularly interesting, and the pathologic picture described deserves careful study from clinical and etiologic viewpoints. The most important points for discussion are: First, is the described pathologic picture the expression of an etiologic entity? Second, if so, is the etiologic factor that of beriberi?

The chief diagnostic points in the pathology are dilatation and hypertrophy of the right heart, anasarca and congestion of internal viscera, and congestion of the lungs. Most of these findings, as the authors state, may be explained by the condition of the lungs. The described lesions in these latter organs are somewhat similar to those often seen in acute bronchitis in infants, especially when autopsy has been delayed for several hours. Acute respiratory troubles are very common in infants in this country (16 of the authors' series of 219 died of pneumonia), and in the further study of these cases these should be carefully considered, both from the clinical and pathologic side. The anatomic picture of beriberi in the adult is not very characteristic, and in most instances an anatomic diagnosis is possible only by exclusion. Beriberi is, of course, a neuritis and histologic studies always show lesions of the nerves, but even these are not of a proved specific nature. The clinical side of the subject is not fully worked out, and usually probably includes other conditions besides the neuritis. Finally, in spite of the recent brilliant work of Fraser and others on the etiology of the disease, the present status of the beriberi question is indefinite and its etiology is not finally

determined. Its position is comparable with that of malaria before Laveran's discovery of the parasite.

In view of this, would it not be better to retain the native names of *suba* or *taon* for the condition described by McLaughlin and Andrews at least until such time as the etiologic agents and clinical picture are made more definite?

The hypothesis that a faulty or disturbed metabolism is the etiologic factor in this group of cases might be open to some criticism, at least in the light of our present knowledge. This is particularly true as to the separation of breast-fed from artificially fed children. Experience in hospital wards and clinics has shown that while practically all Filipino children are fed from the breast, particularly vicious forms of artificial feeding also are at a very early age made to supplement nature's method. Again, it should be mentioned that numerous analyses of mothers' milk taken from the class of people under discussion and analyzed by Bliss, Richmond,[1] Bacon and others of the Bureau of Science have not shown abnormalities in the composition of the milk which would indicate that it is responsible for a sudden acute, fatal disease.

I would also like to call attention to the fact that tuberculosis is extremely prevalent among nursing Filipina mothers.

Dr. Francis Clark, medical officer of health, delegate from the Government of Hongkong.—I believe this to be one of the most interesting papers which has been read. The question of infant mortality frequently arises in the Far East, and at least two-thirds of the natives born die within the first year. The entity described by Doctors McLaughlin and Andrews would suggest the formation of commissions and of systematic inquiries and researches in order further to elucidate the source of infant mortality. Although such commissions have existed, up to the present time they have borne but little fruit. Most careful research has failed to disclose the real causes of death in many instances. I believe the suggestion that many of these deaths are nutritional to be an original one and it opens a new field for research. I believe it to be premature to term this pathologic entity "beriberi." We may regard the disturbances as nutritional without designating them as beriberi. They are perhaps due to nonsufficiency of phosphorus or salts. Doctor Musgrave tells us that in general the milk furnished by the nursing mother is much the same as it is in more temperate countries, but perhaps the content of the milk in salts, and their nature, should be more fully investigated.

Dr. Isaac W. Brewer, Medical Reserve Corps, United States Army.—I have to a certain extent investigated the mortality among infants under one year in the Island of Cebu and have found it to be between 16 and 20 per cent. In that island I found a number of children fed

[1] *This Journal, Sec. B* (1907), 2, 361.

entirely by artificial means. They were given coconut milk, *tuba*,[2] and rice water.

Dr. Ham Aron, professor of physiology, Philippine Medical School.—I have considerable experience in the matter of infant feeding from my connection with the various feeding stations established under my supervision by the Department of Public Instruction, especially in the poorest district of the city, namely, Tondo. My observations have led me to conclude that many of the breast-fed children receive rice or tapioca in addition to mother's milk. I have seen cases of the disease termed beriberi by Doctors McLaughlin and Andrews and which the natives designate as *"taon."* The symptom complex seems to me very closely to resemble the condition which Czerny and the German authors describe as "Mehlnährschaden." I learned that many Filipino children, although breast-fed, in addition are overnourished with the carbohydrates derived from rice or tapioca. The milk of Filipina women is poorer in composition than that of European women. Analyses of individual samples made without regard to the time of day, whether the sample is taken before or after meals, or without taking into account all factors, are of no great importance. However, such as we have show the milk of the Filipina mother apparently to be richer in sugar and poorer in fats and carbohydrates than that of a normal European mother.

I was able to produce in dogs a severe disease by overfeeding them with carbohydrates. The disease is especially characterized by a severe œdema. The clinical picture and the autopsy findings in my experimental dogs in many respects resemble the condition which has been termed beriberi in infants.

Dr. H. Campbell Highet, principal medical officer local government of Bangkok, delegate from His Imperial Majesty's Government of Siam.—The paper which has been read is one of the most interesting which has been given at this session. Only a very minute proportion of the people dying in the East are attended by qualified physicians and hence the usual statistics concerning the causes of death are misleading. A check upon the records is so difficult to obtain, that the work of Doctors McLaughlin and Andrews is of supreme importance. Their results seem to me remarkable in calling attention to the great frequency of infantile beriberi. Whether the condition is true beriberi or not, I am neither prepared to agree to nor to deny. I would prefer to suspend judgment until further work has been done. The pathologic appearances which have been described are certainly those with which we have become very familiar in the cases of adults dead of beriberi, but that breast-fed infants could suffer from beriberi is probably a new fact to

[2] *Tuba*, a fermented juice derived from the cut flower of the coconut palm. On standing for a day or two, it ferments and forms an intoxicating drink.

most of us. This brings me to the remarkable figures which have been given of the comparative death rate between breast-fed and artificially fed infants. Surely there is a flaw somewhere in the statistics, for the proportion of 70 per cent of deaths in breast-fed infants is totally at variance with that shown us by the history of the world since the days of Adam. I was glad to hear that the authors of the paper did not carry this question to its logical conclusion, and advise artificial feeding. The flaw in the figures is probably due to incorrect information with regard to the feeding of the infants. As had already been pointed out by one of the previous speakers, very few of the native infants are entirely breast-fed. This is also true in Siam. As a rule, at the third month, but often earlier, in the latter country mothers are in the habit of supplementing their milk by feeding their children on softboiled rice, uncooked bananas, and frequently on other articles of diet of a far more indigestible nature.

I have always been of the opinion that the high death rate among infants is the result of digestive troubles brought on by all the mixtures rammed down these poor infants' gullets, but that the condition produced might be beriberi is a new consideration to me. However, the subject of infant mortality is of supreme importance in any country. It is in Siam and no doubt in the Philippine Islands as well, so that I hope the mother will always be kept to the fore in the work of this association.

Dr. Victor G. Heiser, Director of Health for the Philippine Islands professor of hygiene, Philippine Medical School, Manila, P. I.—This paper is a most valuable contribution on the subject of infant mortality in the Philippines, and is, I believe, in spite of all that has been said and written upon this question, the first scientific work which has been done for the purpose of ascertaining the actual cause of death in a series of cases.

I feel that a statement should be made in regard to the statistics relating to naturally and artificially fed infants. Several years ago the Bureau of Health was requested to collect statistics showing whether the deaths which occurred among infants under one year of age were in cases which had been naturally or artificially fed; at that time I demurred because of the improbability of obtaining reliable figures of this nature by inquiries made by a clerk. It is well known to those who have experience in the Philippine Islands that almost from birth it is the custom to give rice, potatoes, and other solid food to children that are nursing. Anyone who has the least doubt in regard to the accuracy of this statement can easily satisfy himself by going through any native town and observing the children, and in a comparatively few observations he can witness mothers engaged in this practice. Furthermore, it is characteristic of the more ignorant classes to reply to questions of this nature in the manner in which they believe an answer is desired,

so that when death certificates are presented and the family is asked whether the child had been artificially fed or breast-fed the mother would probably reply breast-fed, and perhaps deny other forms of feeding if she thought such an answer to be wanted.

One year ago, however, at the urgent request of a number of physicians of the city, I reluctantly gave instructions to have statistics of this kind gathered and as you perceive, according to them, 70 per cent of naturally fed children have died, which fact alone indicates that from our present knowledge of infant feeding these figures are not likely to be correct. I desire to disclaim all responsibility on behalf of the Bureau of Health for the statistics which have been used in the paper just read covering this particular point. However, in view of the showing just made, an investigation will be made on this point which will be based upon actual observation of infants in their homes.

The authors of this paper are to be congratulated upon the excellent manner in which they have presented this matter and the concise and clear arrangement of their statistics. They have made a valuable contribution concerning the cause of infant mortality in the Philippines, and with the data brought to light by Doctor Aron we should soon be in position to take effective steps to reduce the mortality.

Dr. Paul Clements, of the Bureau of Health, Manila, P. I.—I have been engaged to a certain extent on the other end of the work described by Doctors McLaughlin and Andrews. About 30 cases in which the pathologic diagnosis was infantile beriberi were sent to the morgue from my station, and it fell to my lot to collect the clinical histories. With one or two exceptions the patients all were babies under three months of age and the large majority were between one and two months. In the course of this work the clinical picture of the disease has assumed quite as much distinctness in my mind as Doctor Andrews says the pathologic picture has in his; and I have no doubt whatever that the disease is a distinct clinical and pathologic entity. My own investigations among the people with regard to the age at which mixed feeding of Filipino babies is commenced leads me to agree more fully with Doctor Highet than with any of the other gentlemen who have spoken on this subject. I have rarely, not often, seen babies younger than three months who were breast-fed and at the same time received only a negligible quantity of other food.

With regard to the name by which we still designate this pathologic entity, it seems that the authors of the paper have merely followed the nomenclature adopted by the first Japanese observer. The condition has been clearly recognized by some Filipino practitioners, and I believe Doctor Albert was the first to call the attention of the profession to its existence. The local name "*taon*" might perhaps be accepted.

Dr. Antonio G. Sison, second assistant resident in clinical medicine, Philippine Medical School, Manila, P. I.—The name malnutrition sug-

gested by Doctor Aron as the cause of high infant mortality instead of beriberi, I think to be more reasonable because of the following: First, the high infant mortality is for the greater part among the poorer class. The women of the working class are at work most of the time and can not feed their babies properly and so are compelled to leave their children to the care of ignorant brothers or sisters, who give the infant anything they have at hand whenever it cries. Second, even if the babies are fed solely on the breast, the mother, because of her ignorance, usually gives her breast to her baby every time it cries, and this practice, in time, leads to malnutrition. Third, the poorer class of women who have been delivered in the hospitals of Manila, and there are taught to feed their infants properly, leave the hospital when they have recovered and never return for the treatment of the child if it becomes ill. For the reasons stated above, I believe the high infant mortality in the Philippines to be due to malnutrition, and this high mortality can be decreased by bettering the conditions of the poorer class socially and above all by educating them with respect to the care of their children.

Dr. Fernando Calderon, professor of obstetrics, Philippine Medical School, Manila, P. I.—It is doubtless true that babies purely breast-fed up to three months die of a disease called *"taon,"* possibly infantile beriberi. I have patients who have borne children all of whom were purely breast-fed, and nevertheless these children died at the age of three months. For this reason, I think that the establishment of institutions like the Gota de Leche [3] will be a great factor in decreasing the high infant mortality. Instead of having one such institution, we should establish ten to twenty in our city. Of course, the founding of charities of this class must be associated with the education of women of the poorer classes in the proper feeding of their children.

Dr. Vernon L. Andrews, Bureau of Science, assistant professor of pathology and bacteriology, Philippine Medical School, Manila, P. I.—In these cases we have a pathologic entity, and while we do not positively maintain it to be moist beriberi, yet we know of no better name for the condition, and if one can be suggested we will be glad to adopt it. We believe that this pathologic entity to be brought about by a nutritional disturbance. It is similar in many respects to moist beriberi in adults. Nearly all of these infants we have examined were under two and one-half months of age, and while this does not preclude the mothers from having given them a mixed diet, we feel that the giving of extraneous material is a negligible quantity in such early infancy. Furthermore, the majority of these infants were children of beriberic mothers, that is, of women in whom symptoms of beriberi were present.

[3] An institution in Manila, established in 1904, devoted to providing pure milk at a small price to the poorer classes.

As to malaria being a causative factor in producing convulsions in these cases, I can only say that the blood was not examined, as the infants were not seen clinically. In one or two instances I found an enlarged spleen and a liver of a chocolate-brown color.

Dr. Allan J. McLaughlin, Assistant Director of Health, assistant professor of hygiene, Philippine Medical School, Manila, P. I.—I wish to correct a false impression which Doctor Musgrave has gathered from our paper. We have never dogmatically said that this disease is beriberi. By referring to our paper it will be noted that we belive we have demonstrated a pathologic entity responsible for many deaths of infants and for which we know of no better term than moist beriberi. Perhaps Doctor Musgrave can suggest a better one; if so, we shall be happy to substitute his diagnosis, tentatively at least, until the question of name is ultimately decided.

In regard to his statement that there are no breast-fed children, strictly speaking, in the Philippines, I desire to state that we are perfectly cognizant of the very common practice among the poorer classes of Filipino mothers of giving young children a variety of solid food in addition to the supply drawn from the breast; but this does not constitute artificial feeding, and the fact remains that the bulk of the children of the Filipino poor are mainly breast-fed. The majority of the deaths from the disease in question, which we tentatively term beriberi, occur in infants under three months of age.

The pernicious custom of giving solid food to the children is not usually practiced until after the infant is three or four months old. We admit, as Doctor Musgrave has suggested, that many Filipina mothers are tubercular, but this condition can only be considered as another factor in the poor nutrition of the Filipina mother. Tuberculosis is not hereditary and a diligent search for tubercular lesions in the cases under discussion produced negative results.

DISCUSSION ON THE PAPER, "UNSOLVED HEALTH PROBLEMS PECULIAR TO THE PHILIPPINES," BY DOCTOR HEISER.

Dr. H. Campbell Highet.—I have listened with great pleasure to Doctor Heiser's paper and at the same time with the deepest sympathy. Those of us who are engaged in administering sanitary measures in the Tropics all know the extent of the task set us and all of us have suffered from the want of funds. In fact we are often apt to think that our respective governments show but little sympathy with us, so small is the appropriation allowed for sanitary work, but, as Doctor

Heiser truly says, it is for us to do the best we can with our limited means and to discriminate carefully along what lines we should work.

Doctor Heiser has submitted an extended programme for the future, a programme which would really prove to be a Herculean task, but it is a good thing to see that he is hopeful of ultimate success. It is well, however, that those of us who have had the privilege of seeing something of the actual working of the Bureau of Health should look back and consider what had already been done to improve the sanitary condition of the people. In a matter of nine years the American Government has worked miracles, and I am certain that my fellow-delegates will join with me in heartily congratulating the Bureau of Health, and especially Doctor Heiser, on their great work.

Dr. J. M. Atkinson, principal medical officer, Hongkong, delegate from the Government of Hongkong.—I have much pleasure in seconding Doctor Highet's remarks congratulating Doctor Heiser on what has already been accomplished by the Bureau of Health. As far as I am aware, there is as yet no other example in the history of the world where such effectual measures have been taken to improve the sanitary condition of the subject native races those as which have been undertaken by the American Government since its occupation of these Islands, a period of less than twelve years.

To protect 6,000,000 people out of a total of 9,000,000 from smallpox is a wonderful piece of work. With regard to the city of Manila and its freedom from malarial fever, it appears to me that this freedom may be only temporary, as were the Filipinos to become infected with malaria, all the white people living in the city would be liable to infection. Mosquitoes capable of transmitting the malarial parasite are already present and extensive areas of swampy land exist.

I would suggest the advisability of gradually filling in these swamps as funds permit, as it is impossible effectually to drain them, since much of the area is below the level of the high tides prevailing. The filled-in swamps could be utilized for building sites, etc., and thus prove remunerative.

DISCUSSION ON THE PAPER, "THE PARTHENOGENESIS OF THE FEMALE CRESCENT BODY," BY DOCTOR NEEB.

Dr. E. R. Stitt, surgeon, United States Navy, associate professor of medical zoölogy, Philippine Medical School.—I have frequently, upon questioning others, found that they had never observed the phenomenon of parthenogenesis in connection with the benign tertian parasite, the species upon which Schaudinn made his observations.

In careful and repeated examinations of the blood of the large number

of marines at the Naval Hospital in Washington, who had never been infected in Panama, and in which blood there were large numbers of gametes, no appearances which would indicate parthenogenetic division were noted. Within the past few months I have observed at Cañacao Naval Hospital a case in which there had been repeated attacks of malaria covering a period of one year. Upon examining the blood of this individual, numerous macrogametes were observed but no schizonts. A few days later parasites resembling Schaudinn's diagram of parthenogenesis were observed, and a few days later the man had a typical malarial paroxysm and in his blood numerous nonsexual macrogametes were visible, and as the microscopic appearance of the parasites showing sporulation, differed from that of the ordinary merocyte I was convinced that the phenomenon I observed was that of parthenogenesis of *Plasmodium vivax*.

Dr. H. M. Neeb.—I wish to add to my paper that fig. 5 is given in the work of Professor Ruge. The difference between fig. 5 of my paper and Ruge's is that Ruge gives a figure of a red blood cell in which are to be seen two parasites—one is a segmenting tertian parasite, the other next to it is a gamete. But in my plate a protoplasmic band combines both halves of this parasite. I can also absolutely state that this figure represents one parasite, and not a mixed infection of the red blood cell.

DISCUSSION ON THE PAPER, "MALARIAL FEVER DURING THE PUERPERIUM," BY DOCTOR ATKINSON.

Dr. Aldo Castellani, professor of tropical medicine and lecturer on dermatology, Ceylon Medical College, delegate from the Government of Ceylon.—I quite agree with Doctor Atkinson that in a malarial country quinine should be given to pregnant women. This is the rule I always follow in those districts of Ceylon where there is malaria, giving 5 grains (0.3 gram) every day and 10 grains (0.6 gram) once a week. Lately I have frequently used euquinine, which is said to have less action on the uterus than the ordinary preparation of quinine.

I also agree with Doctor Atkinson when he says that in malignant malaria larger doses of quinine must be given. It is probable that in a temperate zone 1 gram (15 grains) a day is sufficient to stop an infection with malignant malaria, but in tropical countries, like East Central Africa and Ceylon, I find that much larger doses must be given. In some cases 2 to 2.6 grams 30 or 40 and more grains a day are often necessary.

Doctor Atkinson.—I was not aware that euquinine had less effect on the uterine muscles than quinine. We always give euquinine to children

suffering from malaria because it has no taste and hence is easier to administer.

I encountered a number of cases of malaria in the year 1889, after the great rainfall which we had in Hongkong. You probably have all heard of this downpour during which 30 inches (900 millimeters) fell in twenty-eight hours. As a natural result much malaria developed at that time, and at least 12 malignant cases came to the Government Civil Hospital.

Dr. J. M. Phalen, captain, Medical Corps, United States Army, member of United States Army Board for the Study of Tropical Diseases as they Occur in the Philippine Islands.—While recognizing the necessity of large doses of quinine in exceptional cases, I wish to call attention to a danger which is present in administering such large amounts. It not infrequently happens that impaired vision supervenes, dating from the time of the administration of quinine in doses which are not to be regarded as excessive. I am not referring now to the quinine amaurosis coming on suddenly and causing more or less temporary blindness, but to a gradual diminution of visual acuity apparently due to the action of quinine on the optic nerve. I am strongly of the opinion that quinine should be given in the minimum dosage that will control the disease.

Doctor Atkinson.—In reply to Doctor Phalen's statement concerning the occurrence of amaurosis after the giving of quinine, all I can say is that in my experience of practically hundreds of cases in which the drug has been given, I have never seen this result.

DISCUSSION ON THE PAPER, "SOME CLINICAL ASPECTS OF MYCETOMA, AN UNUSUAL FORM OF CALLOSITY COMPLICATING IT," BY MAJOR HOOTON.

Dr. J. M. Atkinson.—During my stay in Hongkong I have seen three cases of mycetoma, or Madura foot—one in a Chinaman, the other two in Indians. The fact that we occasionally see these diseases in Hongkong is due to the cosmopolitan nature of the population of that port.

I would like to ask Major Hooton what treatment he adopted in case number 2, and if scraping will cure the disease?

In Hongkong the Chinese frequently suffer from a carinous affection called "by-head." In this affection all the bones of the face and head become enormously enlarged. It has occurred to me that this may be due either to mycetoma or actinomycosis.

Doctor Musgrave.—I enjoyed hearing Major Hooton's paper. The subject of mycetoma is a very large one and I think that we will

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Doctor Musgrave.—I enjoyed hearing Major Hooton's paper. The subject of mycetoma is a very large one and I think that we will

bring order out of chaos only by systematic study. The variations in culture of *Streptothrix* are greater than in any other organism. It must be remembered that none of these *Streptothrix* infections are inflammatory processes. They are destructive without much inflammation.

As to actinomycosis, I think perhaps it had better be included in the list of the *Streptothrix* infections.

Doctor Atkinson.—In connnection with *Streptothrix* infections, I should like to ask Doctor Musgrave and Major Hooton if there is any similar infection in horses in their respective countries? We have something approaching it in China.

Doctor Musgrave.—So far as I know there has been only one case reported in a native animal of the Philippines. I have seen it in Australian and American horses.

Maj. A. Hooton, I. M. S., Rajkot, Kathiawar, India, delegate from the government of India.—I will state in reply to Doctor Atkinson's question that the treatment I adopted in that particular case was the removal of the growth.

As regards the question of *Streptothrix* infection in animals, I made inquiries in Bombay and was told that perhaps two or three cases of mycetoma in animals are encountered in a year.

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